



Reactive vs proactive aggression: A differential psychobiological profile? Conclusions derived from a systematic review

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Highlights

- Reactive aggression is characterized by emotional lability.
- Proactive aggression is driven by low emotionality.
- Doubts regarding dichotomic or complementary nature.
- Heritability accounted for 45% of the explained variance in both types of aggression.
- Opposite pattern for allelic variants serotonin transport and monoaminoxidase.

- Brain analysis overlap in prefrontal cortex, amygdala and temporal cortex.

Abstract

Introduction

Scholars have established subcategories of aggressive behavior in order to better understand this construct. Specifically, a classification based on motivational underpinnings makes it possible to differentiate between reactive and proactive aggression. Whereas reactive aggression is characterized by emotional lability, which means it is prone to impulsive reactions after provocation, proactive aggression is driven by low emotionality and high levels of instrumentality to obtain benefits. Some authors have conceived these two types as having a dichotomous nature, but others argue against this conceptualization, considering a complementary model more suitable. Hence, neuroscientific research might help to clarify discussions about their nature because biological markers do not present the same biases as psychological instruments.

Aim

The main objective of this study was to carry out a systematic review of studies that assess underlying biological markers (e.g., genes, brain, psychophysiological, and hormonal) of reactive and proactive aggression.

Methods

To carry out this review, we followed PRISMA quality criteria for reviews, using five digital databases complemented by hand-searching.

Results

The reading of 3993 abstracts led to the final inclusion of 157 papers that met all the inclusion criteria. The studies included allow us to conclude that heritability accounted for approximately 45% of the explained variance in both types of aggression, with 60% shared by both, especially, for overt and physical expression forms, and 10% specific to each type. Regarding allelic risk factors, whereas low functioning variants affecting serotonin transport and monoaminoxidase increased the risk of reactive aggression, high functioning variants were associated with proactive aggression. Furthermore,

brain analysis revealed an overlap between the two types of aggression and alterations in the volume of the amygdala and temporal cortex. Moreover, high activation of the medial prefrontal cortex (PFC) facilitated proneness to both types of aggression equally. Whereas stimulation of the right ventrolateral (VLPFC) and dorsolateral (DLPFC) reduced proneness to aggression, inhibition of the left DLPFC increased it. Finally, psychophysiological and hormonal correlates in general did not clearly differentiate between the two types because they were equally related to each type (e.g., low basal cortisol and vagal variability in response to acute stress)

Conclusions

This study reinforces the complementary model of both types of aggression instead of a dichotomous model. Additionally, this review also offers background about several treatments (i.e., pharmacological, non-invasive brain techniques...) to reduce aggression proneness.



Previous

Next



Keywords

Brain; Drug; Proactive aggression; Psychobiology; Reactive aggression

1. Introduction

Scholars have made important efforts to understand the etiology of human aggressive behavior. Accordingly, the greater our knowledge about the mechanisms underlying it, the greater our ability to develop effective treatments to reduce it and better strategies to prevent it. A widely employed method to reduce the complexity of a broad concept like aggression consists of building subcategories of it and establish their differences ([Martín-Ramírez and Andreu, 2006](#)). An extensive classification might be based on motivational underpinnings or motives for aggressive behavior, making it possible to differentiate, for example, between reactive and proactive aggression ([Martín-Ramírez, 2010](#); [Moya-Albiol and Romero-Martínez, 2020](#)).

Whereas reactive aggression is characterized by negative affect and/or emotional lability (e.g., often called ‘hot-blooded’), which makes the person prone to impulsive reactions after provocation, proactive aggression tends to be driven by low emotionality

(e.g., ‘cold-blooded’ or lack of emotional arousal) and high levels of instrumentality to obtain benefits and/or rewards in the absence of provocation (Dodge, 1991, Dodge and Coie, 1987).

Apparently, some authors have described them as two opposite forms of aggressive behavior with a dichotomous nature (Fite et al., 2012; Hoffer et al., 2018; Meloy, 2006; Vitaro and Brendgen, 2011). This dichotomy was mainly based on completely different emotional and cognitive processes underlying each type of violence (e.g., cognitive biases, emotional deficits, closeness to psychopathic traits, etc.). Other scientists have argued against this conceptualization. They defend the usefulness of conceptualizing them as a continuum where each extreme is represented by a type of aggression, given that the two forms of aggression are not mutually exclusive and frequently co-occur (Mäki-Marttunen et al., 2019; Raine et al., 2006; Walters, 2005). This representation of aggressive behavior has been supported by empirical research employing self-reports, which frequently reveal a stronger statistical link between two scales (e.g., correlation coefficients higher than .50) (Polman et al., 2007).

As previously explained, most of the research assessing differences has been based on a psychological perspective. Therefore, one way to provide enough background and, in turn, clarify this question would be to introduce an interdisciplinary approach. Specifically, neuroscientific research might add complementary information to the classical psychological perspective. Furthermore, neuroscientific disciplines include biological markers such as allelic gene variants, brain circuits, and psychophysiological markers, among others, which do not present the characteristic biases of psychological instruments.

Considering the above, and to provide a systematic synthesis of the scientific literature that has analyzed biological correlates of reactive and/or proactive aggression in humans, the present study first describes the main findings on heritability and genetic markers (e.g., allelic risk factors). Second, with the main purpose of understanding brain correlates, we describe the results of brain morphological analysis related to each type of aggression, followed by functional and activation analysis and results of experiments that include non-invasive brain stimulation techniques and, finally, the effects of drug administration. Afterwards, we summarize the psychophysiological correlates (e.g., cardiorespiratory and electrodermal markers) and, finally, the hormonal markers of the above-mentioned aggression styles. Thus, considering the existing data so far, we have tried to answer whether neuroscientific markers might allow us to distinguish between

the two forms of aggression, supporting a dichotomous or, conversely, complementary and not mutually exclusive model.

2. Methods

2.1. Search strategy

To conduct this systematic review, we followed the recommendations stated in the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines in order to increase the quality and reliability of this review ([Moher et al., 2009](#)). Consequently, we employed five digital databases to carry out this study: PubMed, PsycINFO, Scopus, Web of Knowledge, and Cochrane Library (OSF: Registration DOI: 10.17605/OSF.IO/97ZXV). It should be highlighted that several databases provided the opportunity to apply filters to conduct the research, and so we specifically removed, when possible, “books”, “PhD Thesis, graduate theses and dissertations”, “case reports”, “animals”, and “review”, and we selected the language: “English and/or Spanish” because of the reviewers’ language expertise and ‘human research’. However, we decided not to apply date restrictions to cover as many studies as possible.

Searches in these digital bases were complemented by hand searching for additional articles that met the eligibility criteria and were not included in the aforementioned digital databases. For this purpose, we employed ResearchGate and Google Scholar.

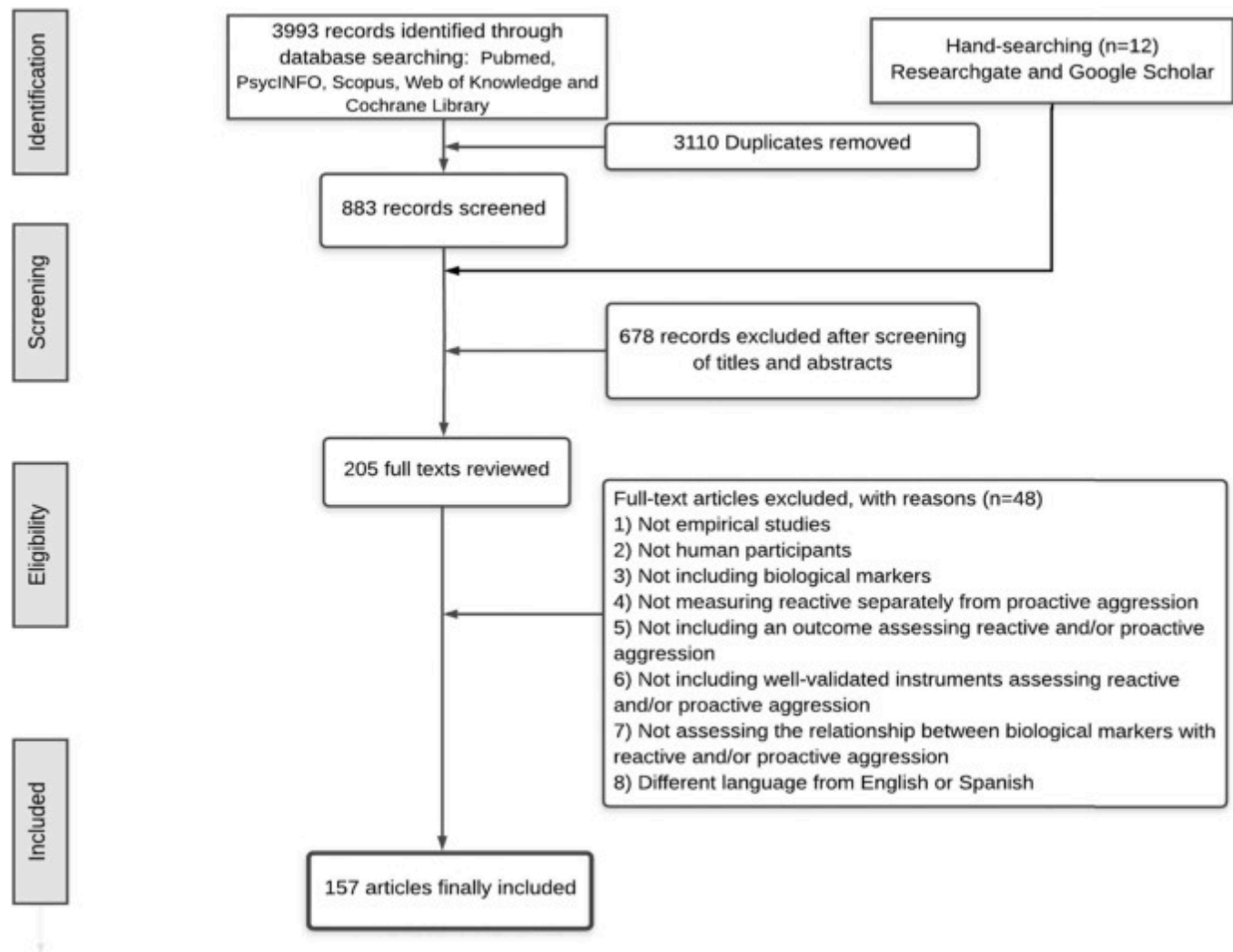
The search for all published studies was performed from January to June 2021.

The algorithm employed to conduct this review was: ([*proactive* **OR** *reactive*] **AND** [*violence* **OR** *aggression*]) **OR** ([*Point Subtraction Aggression Paradigm* **OR** *Taylor Aggression Paradigm* **OR** *Anger Infused Ultimatum Game*]) **AND** [*gene* **OR** *brain* **OR** *serotonin* **OR** *hormone* **OR** *heart rate* **OR** skin conductance **OR** *autonomic functioning*]).

2.2. Inclusion criteria

We formulated our selection criteria according to recommendations by [Higgins et al. \(2019\)](#) for conducting systematic reviews. Thus, we finally selected the manuscripts that met all these criteria: (a) empirical studies (except for case reports); (b) studies that explicitly involved human participants; (c) normative or clinical populations; (d) included psychobiological markers (e.g., genes, brain correlates, neurotransmitters, psychophysiological and hormones); (e) measured reactive aggression separately from proactive aggression; (f) assessed the relationship (i.e., correlational, regression analysis,

direct comparison...) between reactive and/or proactive aggression and biological parameters; (g) were written in English or Spanish (PRISMA flow diagram; Fig. 1).



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Fig. 1. PRISMA flowchart of literature search with reasons for exclusion.

2.2.1. Exclusion criteria

We also specifically checked for studies that measured aggression with laboratory tasks if they included a total score for each type of aggression (i.e., total punishment applied to opponent, percentage of aggressive responses across trials...). If manuscripts assessed aggressive behavior during tasks (e.g., decision phase, outcome phase...), but without providing an outcome, they were removed from this review. Additionally, studies where researchers did not explicitly mention that they measured reactive and/or proactive aggression were not included (e.g., defensive aggression but not reactive). Furthermore, it was important to include well-validated instruments to measure both types of

aggression. Lastly, studies that did not provide enough demographic information were not included.

2.3. Quality assessment

Although the quality of a systematic review and a meta-analysis might be guaranteed by selecting only research with robust experimental designs, such as double-blinded randomized controlled trials, we assumed that much of the important research for this review would present non-robust designs (i.e., cross-sectional, correlational, open clinical, etc.). Hence, we finally decided not to establish the type of design as a selection criterion, except for case studies.

Due to the broad heterogeneity in the studies' methodological characteristics, we considered it particularly interesting to assess the quality of the studies by providing a [supplementary table](#) summarizing key points of their designs. This would help to understand the potential risk of bias when interpreting the data. Based on previous literature establishing quality criteria for non-randomized and randomized studies, we included eight statements that would be answered with a dichotomous answer (Yes/No) indicating whether the criterion was met or not. Based on the answers we indicated whether studies presented a low, moderate, or high-risk bias. (See [Supplementary Table 1](#)).

Supplementary Table 1.

Summary of design quality for included studies.

Based on previous scientific literature in this field, we considered it appropriate to define the following statements:

2.4. Sample

1a) Specified inclusion criteria for each group.

1b) Randomization or Pseudo-randomization.

1c) Offered enough information about experimental mortality.

2.5. Procedure

2a) Blinded (e.g., double, or simple).

2b) Employed reliable instruments (except one of them).

2.6. Results

3a) Matched groups (e.g., number of participants per group...).

3b) Covariate potential confounding variables.

3c) Replicable results (e.g., offered enough background and correctly justified).

2.7. Data extraction and analysis

Article selection was carried out by two scholars, with an interrater agreement above 90%. Any possible discrepancies between the two raters were discussed, and they clarified whether critical points met the inclusion criteria by consulting the third author of the manuscript.

3. Results

3.1. Selection of studies

From a total of 3993 publications retrieved from the search of the digital databases and 12 from hand-searching, 3110 articles were removed because they were duplicated, and 687 were excluded based on the title or abstract because they were not relevant to the main objective of this review. Hence, 205 full-text articles were screened for eligibility, with 157 finally being included (see flowchart described in [Fig. 1](#)).

3.2. Genes

3.2.1. Twin studies

With regard to the quality of the studies included in this block, it has been suggested that it would be appropriate to include more than 600 pairs of twins to conduct twin studies with enough statistical power ([Martin et al., 1978](#)). Accordingly, only one of the studies included presented a sample size larger than 600 pairs of twins ([Baker et al., 2008](#)). The rest of the studies presented sample sizes ranging from 900 to 700 participants ([Fullerton et al., 2019](#), [Orri et al., 2020](#), [Paquin et al., 2014](#), [Paquin et al., 2017](#), [Tuvblad et al., 2009](#)), and, finally, one research study included less than 400 participants ([Brendgen et al., 2006](#)). However, it should be highlighted that all these studies employed well-known self-reports validated in several languages (for further details, see [Supplementary Table 1](#)).

The analysis of twin studies with both genders concluded that the amount of variance explained by genetic factors (i.e., heritability) in reactive and proactive aggression ranged from 26% to 45%, with approximately 40% of the rest of the variance explained by nonshared environmental factors and 10% by shared environmental factors (Brendgen et al., 2006, Paquin et al., 2014, Tuvblad et al., 2009). Curiously, they also pointed out that approximately 64–76% of these genetic factors seemed to be shared in both types of aggression, specifically, the forms associated with the physical expression of aggression (Brendgen et al., 2006, Paquin et al., 2017). Nevertheless, only a small percentage of genetic factors (smaller than 11%) seemed to be specific to each type (Brendgen et al., 2006). Curiously, if only the impulsive component of reactive aggression was considered, genetic factors accounted for 90% of the explained variance (Orri et al., 2020).

Gender is differentially related to heritability in both types of aggression. In fact, these factors seemed to be more important in men (e.g., 38% for reactive and 50% for proactive aggression) than in women. Conversely, in women, shared and non-shared environmental influences were important in both reactive and proactive aggression (Baker et al., 2008).

As mentioned in the previous paragraphs, for both types of aggression, nonshared environmental factors were particularly important (Brendgen et al., 2006). In fact, one study indicated that a previous history of traumatic brain injuries (TBI) explained differences in reactive and/or proactive aggression during maturation processes in children (Fullerton et al., 2019). In this regard, adolescents of both genders who suffered a TBI earlier in their lives presented higher reactive aggression levels, but lower proactive aggression levels.

3.2.2. Risk alleles

Of a total of 14 manuscripts included in this section, seven articles found an initial significant association between allelic variation and reactive and/or proactive aggression. Furthermore, of those that did not report a significant initial association, after considering additional variables, five of them reached significance (for further details see Table 1).

Table 1. Main sociodemographic characteristics and details about the participants in each study, the main results, and the assessment methods used.

Authors	Sample size	Age, gender and type of group	Genetic variable	Aggression assessment	Statistical analysis; design study	Signi resul (dire assoc
Twin studies						
Baker et al. (2008)	1219	9.60 ± 0.60;; 48,7% ♂ 51,3% ♀; normative; 37.5% Hispanic, 26,6% Caucasian		RPQ	Twin study (cross- sectional) (comparative study)	p < .0
Brendgen et al. (2006)	366	From 5 to 60 months; 50% ♂ 50% ♀ normative; 84% European descent	–	ABTC	Twin study (longitudinal) (comparative study)	p < .0
Fullerton et al. (2019)	882	From 9 to 20 years; 43% ♂ 57% ♀ normative; 35% White and 30% Hispanic	–	RPQ	Twin study (longitudinal) (comparative study)	p < .0
Orri et al. (2020)	862	From 6 to 12 years; 41.2% ♂ 58.8% ♀ -	–	ABTC	Twin study (longitudinal) (comparative study)	p < .0
Paquin et al. (2014)	667	From 6 to 12 years Non-specified normative; 84% European descendent	–	ABTC	Twin study (longitudinal) (comparative study)	p < .0
Paquin et al. (2017) (same data 2014)	667	From 6 to 12 years Non-specified normative; 84% European descendent		ABTC	Twin study (longitudinal) (comparative study)	p < .0

Authors	Sample size	Age, gender and type of group	Genetic variable	Aggression assessment	Statistical analysis; design study	Significance result (direction of association)
Tuvblad et al. (2009)	874	From 9 to 14; 50% ♂ 50% ♀; normative; -	–	RPQ	Twin study (longitudinal) (comparative study)	p < .0
Genetic polymorphisms or gene-sets with self-reports						
Fragkiadaki et al. (2019)	323	From 13.00 ± 0.42–18.00 ± 0.42; 56% ♂ 44% ♀; normative; majority European ancestry	Oxytocin receptor gene: rs1042778, rs2254298, rs53576, rs4686302, rs237915 polymorphisms	RPQ	Regression analysis (longitudinal study)	No (p > .05)
Lazebnik et al. (2020)	35	20.03 ± 2.30; 100% ♂ normative; Russians	SNP: rs1062535; rs13381941; rs1051922	RPQ	ANOVA (comparative study)	Yes (p < .05)
Luppino et al. (2014)	141	7.29 ± 3.14; 72% ♂ 28% ♀ normative; 100% White	Polymorphism rs35369693 located on the AVP 1B receptor	ABTC	One way ANOVA (comparative study)	Yes (p < .05)
Peeters et al. (2020)	85	20.09 ± 0.259; 100% ♀ normative; -	5-HTTLPR S and L polymorphisms	RPQ TAP	Univariate ANOVA (comparative study)	No (n.s.) Yes (p < .05)
Van Dongen et al. (2020)	395	23.08 ± 3.38; 36% ♂ 64% ♀ normative; Caucasian	483 genes (e.g., dopaminergic, serotonergic, and neuroendocrine)	RPQ	The weighted Stouffer's Z method	Non-replicable

						Signi resul (dire assoc
Authors	Sample size	Age, gender and type of group	Genetic variable	Aggression assessment	Statistical analysis; design study	
Weidler et al. (2019)	59	24.86 ± 3.92; 100% ♂ normative; -	OPRM1 polymorphism (G+ and G-)	RPQ TAP	MANOVA(comparative study)	No (p No (n repor
Zhang et al. (2016)	1433	12.32 ± 0.49; 53.8% ♂ 47.2% ♀; normative; 97.6% Chinese Han	MAOA T941G and COMT Val158Met polymorphisms	RPQ	Regression analysis (comparative studies)	No (p
Cherep kova et al. (2015)	565	From 19–64; 100% ♂ convicted men; Caucasian	DRD4 48-bp VNTR polymorphism	Psychiatric interview	Fisher's exact test and the Kendall tau test (Comparative study)	Yes (r repor concl basec few s
Hemmi ngs et al. (2018)	290	21 years; 100% ♂ offenders; 80.7% IsiXhosa ethnicity	5-HTTLPR and rs25531 and STin2 VNTR polymorphism	BPAQ	Regression analysis (cross-sectional)	Yes (q
Kolla et al. (2014)	31	37.07 ± 7.70; 100% ♂ offenders and normative; 65% Caucasian	MAOA-VNTR polymorphism	RPQ	Regression analysis (comparative study)	Yes (q
Kueppe r et al. (2013)	239	23.08 ± 3.38; 36% ♂ 64% ♀ convicted men; Caucasian	MAOA-uVNTR genotyping	FPI-R Competitive reaction time-task	ANOVA (Comparative study)	No (n repor No (n repor

Authors	Sample size	Age, gender and type of group	Genetic variable	Aggression assessment	Statistical analysis; design study	Significance result (direction of association)
Van Donkelaar et al. (2018)	501	25.28 ± 4.62; 43% ♂ 57% ♀ convicted men; Caucasian	MDS1 and EVI1 complex locus gene (MECOM) and Vasopressin Receptor 1 A gene (AVPR1A)	RPQ	Weighted Stouffer's Z method	No (p > 0.05)
Genetic polymorphisms or gene-sets with laboratory tasks						
Gallardo-Pujol et al. (2013)	57	22.77 ± 4.37; 100% ♂; normative; Caucasian	MAOA uVTNR	PSAP	Univariate ANOVA (comparative study)	Yes (p < 0.05)
Schlüter et al. (2016)	88	23.5 ± 3.5; 100% ♂; normative; Caucasian	MAOA LPR	PSAP	Correlation analysis (comparative study)	No (p > 0.05)

Note. ABTC: Aggression Behavior Teacher Checklist; BPAQ: Buss-Perry Aggression Questionnaire; FPI-R: Freiburg Personality Inventory; PSAP: Point Subtraction Aggression Paradigm; RPQ: Reactive Proactive Questionnaire; STAP: Social Threat Aggression Paradigm; TAP: Taylor Aggression Paradigm.

3.2.2.1. Reactive aggression

With regard to reactive aggression, normative children of both genders who were carriers of the CG allele of single-nucleotide polymorphism (SNP) rs35369693, belonging to the Arginine Vasopressin Receptor 1B (AVP 1B) gene, presented higher self-reported reactive aggression than carriers of the GG allele ([Luppino et al., 2014](#)). In addition, the CG allele was associated with alterations in prosociality and emotional empathy, thus facilitating proneness to reactive aggression.

Polymorphism STin2 VNTR12 of the SLC6A4 gene, encoding for serotonin transportation, was negatively associated with this kind of aggression (self-reported) in men offenders ([Hemmings et al., 2018](#)) and when measured with the Taylor Aggression Paradigm (TAP)

in normative women (Peeters et al., 2020). That is, lower expression of the SLC6A4 gene might disrupt the transportation of serotonin to recycle it into the neurotransmitter pool, which in turn affects serotonin regulation. These results were partly supported by a later study in normative young women. Even though they failed to report an initial association between the variables, the authors pointed out that self-reported reactive aggression due to internal frustration was related to SLC6A4 and Cyclin-Dependent Kinase-Activating Kinase Complex Subunit genes (Van Donkelaar et al., 2020). However, it should be highlighted that the number of 5-HT transporters significantly vary in different brain structures (Cortes et al., 1988). Therefore, a general increase or decrease in 5-HT levels cannot be assumed.

Other studies focused on the monoamine oxidase A gene promoter (MAOA-LPR). One of them demonstrated that healthy young men who were carriers of the low-activity MAOA-LPR allele presented higher reactive aggression levels (measured with the Point Subtraction Aggression Paradigm (PSAP) or other competitive tasks) than carriers of the high-activity MAOA-LPR allele (Gallardo-Pujol et al., 2013, Kuepper et al., 2013, Schlüter et al., 2016). Finally, normative adolescents of both genders who were carriers of T alleles/TT homozygotes of the MAOA gene or Met alleles of the catechol-O-methyltransferase (COMT) gene exhibited more reactive aggression, specifically if they were exposed to low positive parenting (Zhang et al., 2016). The low activity variants entailed worse functioning of the MAOA and COMT (reducing their enzymatic activity), which affect the efficiency of monoaminergic neurotransmission (e.g., dopamine, noradrenaline, 5-HT, etc.).

3.2.3. Proactive aggression

Regarding risk alleles for proactive aggression, high activity MAOA-LPR allele (Kolla et al., 2014), as well as low functioning alleles (5 R/5 R, 5 R/7 R and 7 R/7 R VNTR) corresponding to the DRD4 gene (Cherepkova et al., 2015), were associated with high self-reported proactive aggression in men offenders. Specifically, high-activity MAOA-LPR supported more efficient transcriptional activity of the MAOA gene. In contrast to efficient neurotransmission systems, 5 R and 7 R allelic variants of D4 dopamine receptors, they might provoke the appearance of less efficient receptors (e.g., deficient effectiveness in binding to dopamine).

Regarding normative and healthy individuals, the high-activity MAOA-LPR allele was also related to self-reported proactive aggression in young adults of both genders (Kolla et al., 2014), which is congruent with the aforementioned results in male offenders. Furthermore, although specific allelic polymorphisms (e.g., rs1042778, rs2254298,

rs53576, rs4686302, and rs237915) related to transcriptional regulation of the oxytocin receptor (OXTR) gene did not explain self-reported proactive aggression, if these polymorphisms were included with delinquent behavior of friends, the interaction partly explained self-reported proactive aggression in adolescents of both genders (Fragkaki et al., 2019). That is, worse functioning of the oxytocinergic system was related to proactive aggression when carriers of these alleles were in risky environments. Moreover, it should also be highlighted that SNP rs2254298 has been extensively studied due to its association with psychopathology (e.g., depression, autism...) and social cognition.

Additionally, there were also related concrete SNP, such as rs1062535 (of the ITGA2 gene encoding for Integrin subunit alpha 2), rs13381941 (of the ESCO1 gene, encoding for Establishment of sister chromatid cohesion N-acetyltransferase-1), and rs1051922 (of the IFNB1 gene, encoding for Interferon regulatory factor-1), in this kind of aggression (self-reported) in young men (Lazebny et al., 2020). These last allelic variants tend to affect cellular morphological structures and the immune system, among others.

3.3. Brain

This section has been divided into four parts. Accordingly, the first part analyzes alterations in the brain's morphology that might explain reactive or proactive aggression. It is followed by the presentation of specific brain structures' activation and functional connectivity of brain circuits and their association with either of the two types of aggression. The third block is dedicated to explaining how stimulation of these brain structures using non-invasive techniques modulates aggression proneness. Lastly, the main results of drug administration for reactive and/or proactive aggression are summarized.

3.3.1. Morphological analysis

3.3.1.1. Reactive aggression

Self-reported reactive aggression was positively associated with right amygdala volume (Farah et al., 2018) and negatively with left amygdala grey matter volume (GMV) in normative young adult males (Bobes et al., 2013). Furthermore, it was also related to an increased volume of the left putamen in normative adolescents of both genders (Yang et al., 2017). In contrast, this type of aggression (self-reported) was negatively related to GMV in the right middle and superior temporal gyrus in violent offenders and young adults of both genders (Hofhansel et al., 2020, Zhu et al., 2019a), and to reduced GMV of

the insula of adolescents of both genders with conduct disorder (CD) and oppositional defiant disorder (ODD), as well as normative adolescents (Naaijen et al., 2020).

3.3.2. Proactive aggression

In the case of self-reported proactive aggression, the volume and density of the amygdala (particularly right sided), posterior cingulate cortex (PCC), and lateral and medial frontal cortex were negatively associated with this type of aggression in CD, ODD, and normative adolescents, and in young adults of both genders (Bobes et al., 2013, Naaijen et al., 2020, Pardini et al., 2014, Yang et al., 2017, Zhu et al., 2019a). In contrast, another study – which only included healthy young adult men – found that the right amygdala volume and left ACC were positively associated with increased proactive aggression (Farah et al., 2018). Moreover, grey matter density of the dorsolateral prefrontal cortex (DLPFC) bilaterally and cortical thickness in the right anterior cingulate cortex (ACC), medial frontal cortex, and inferior and medial temporal cortex were positively associated with this type of aggression in normative adolescents of both genders (Yang et al., 2017, Zhu et al., 2019a).

3.4. Functional analysis

3.4.1. Reactive aggression

3.4.1.1. Brain activation during resting periods or in response to tasks promoting aggression

Regarding the relationship between reactive aggression and brain activation, a study assessed whether this type of aggression (self-reported) was related to brain activation during a resting period (before undergoing an fMRI task). The authors concluded that it was positively related to activation of the medial PFC (mPFC) and negatively to the posterior insula and the amygdala in offenders and in non-violent normative men (Siep et al., 2019).

With regard to activation during tasks measuring aggressive behavior, a positive association was found between reactive aggression and reactivity of the PFC, ACC, striatum, and insula in normative young adults of both genders to PSAP (Skibsted et al., 2017). Additionally, a study – which included alcohol consumption – concluded that there was a positive relationship between amygdala and ventral striatum activation and reactive aggression during the TAP in normative young adults of both genders, with this association being mediated by alcohol consumption (Gan et al., 2015).

When the sample was divided by participants' sex, reactive aggression (measured with the TAP) was positively associated with activation of the right inferior orbital gyrus/ACC/right rectal gyrus in men, and negatively in normative young adult women (Repple et al., 2018). The rest of the studies only measured brain activation in normative young adult men. Specifically, research revealed that reactive aggression (assessed with the TAP) was associated with increased neural activation in the bilateral insula cortex, left-lateralized motor regions, left frontal lobe, left inferior parietal lobe, bilateral putamen/globus pallidus, left-lateralized thalamus and caudate, and cerebellum (Dambacher et al., 2015a, Emmerling et al., 2016). However, high reactive aggression during PSAP was related to a low dopamine (DA) intrinsic synthesis capacity in the midbrain, caudate nucleus, and putamen (Schlüter et al., 2013) and a higher magnitude of the N2 component in the PFC in response to the TAP (Chen et al., 2019).

Lastly, the comparison of young adult men with anger management problems with controls in response to the PSAP revealed that, specifically, the first group exhibited higher brain activation in the bilateral anterior insula, bilateral putamen, and left inferior frontal operculum, but decreases in activation in the bilateral ventromedial PFC (vmPFC) and right precuneus, in comparison with controls (Gan et al., 2016).

3.5. Brain activation in response to tasks measuring emotional processing

Regarding brain activation in processing emotional stimuli and its association with reactive aggression, one study concluded that there was a negative association between OFC reactivity to angry faces and reactive aggression (measured with the TAP) in healthy young adults of both genders (Beyer et al., 2015). Moreover, another study found a positive association between amygdala bilateral reactivity to angry faces and reactive aggression in normative young women. Furthermore, high reactivity of the left inferior occipital gyrus and bilateral superior temporal gyrus (STG) to emotional faces (angry vs neutral) was related to higher reactive aggression on the Social Threat Aggression Paradigm (STAP) (Buades-Rotger et al., 2016; 2018). In men, a positive association was found between reactivity to emotional stimuli of the right middle frontal/precentral gyrus and left inferior temporal/fusiform gyrus and reactive aggression (measured with the TAP) in healthy young men (Beyer et al., 2014).

Self-reported reactive aggression was negatively associated with activation of the mid and posterior insula and PAG in children of both genders who were watching a video of an individual intentionally harming another individual (Michalska et al., 2016). Lastly,

attenuated activation of the vmPFC in response to a social fairness game has been related to high self-reported reactive aggression proneness in adolescents with ODD and normative adolescents of both genders (White et al., 2016).

3.6. Brain functional connectivity

With regard to resting state functional connectivity (rsFC), a study that only considered women concluded that the rsFC between the basolateral amygdala (BLA) and the superior temporal gyrus was associated with increased reactive aggression in normative young women, whereas BLA and right superior parietal lobule connectivity was related to lower reactive aggression. Furthermore, the higher the changes in functional connectivity between the BLA and medial orbitofrontal cortex (Mofc), the lower the reactive aggression proneness on the STAP (Buades-Rotger et al., 2019).

Regarding functional connectivity in response to tasks, reactive aggression (measured with the TAP and InterTemporal Aggression Paradigm) involved diminished functional connectivity between the left orbitomedial prefrontal cortex (omPFC) and right angular gyrus and bilateral caudate nucleus, as well as reduced functional connectivity between the right omPFC and the right caudate nucleus and between the vmPFC and the frontoparietal network (dorsolateral PFC, superior parietal lobule) and the cingulo-opercular network (dorsal anterior cingulate cortex) in young adults of both genders (Chester et al., 2019, Jiang et al., 2018b).

Moreover, self-reported reactive aggression in aggressive and non-aggressive adolescents of both genders tended to be related to enhanced connectivity between the posterior cingulate cortex (PCC) and the parahippocampus, the left amygdala, and the precuneus, but also reduced connectivity between the right anterior insula and the nucleus caudate (Werhahn et al., 2020).

3.6.1. Proactive aggression

The analysis of brain activation and its relationship with proactive aggression revealed that self-reported proactive aggression was associated with high mPFC and reduced superior temporal sulcus activation during a resting period (pre-laboratory task scanning) in young men offenders as well as in normative men (Siep et al., 2019).

The decrease in the glutamate concentration in the striatal was related to higher levels of self-reported proactive aggression in adolescents of both genders with ODD or CD and normative adolescents (Craig et al., 2019). In addition, this kind of aggressive behavior (measured with the TAP) involved high activation of the right parietal lobe close to the

postcentral gyrus and the right superior temporal gyrus in normative men (Emmerling et al., 2016). Moreover, higher self-reported proactive aggression was positively associated with a higher magnitude of the N2 component of event-related potentials and decision-related negativity in the PFC in young male offenders and in normative males (Chen et al., 2019). Finally, self-reported proactive aggression was negatively associated with activation of the right amygdala during the processing of fearful faces in young people of both genders with CD (Lozier et al., 2014).

With regard to functional connectivity, proactive aggression (self-reported and with the InterTemporal Aggression Paradigm) was related to a reduced functional connectivity between the left PCC and the right DLPFC, right IPL, right mPFC/ACC, and bilateral precuneus, as well as between the dorsal striatum and insula and supplemental motor area, and high functional connectivity between the left DLPFC and the left inferior parietal lobes in young adults of both genders (Chester et al., 2019, Werhahn et al., 2020, Zhu et al., 2019a). Finally, a negative association was found between functional connectivity between the left inferior ventral striatum and the precuneus and self-reported proactive aggression, but only in men with antisocial personality disorder (ASPD) who were carriers of the low activity MAOA genotype (Kolla et al., 2018).

3.6.2. Non-invasive brain stimulation techniques

Of the nine studies that met the inclusion criteria, only four of them presented an initial significant modulation of reactive and/or proactive aggression after applying non-invasive brain stimulation (Chen, 2018; Perach-Barzilay et al., 2013; Riva et al., 2017; Sergiou et al., 2021). Even so, it was necessary to consider ‘session order’, ‘time’, and/or ‘gender’, among others, to reach significance in four other studies. Curiously, most of the research was carried out in healthy young adults of both genders, but only two of them focused on men, specifically in clinical populations (Sergiou et al., 2021, Weidler et al., 2020) (see Table 2).

Table 2. Main sociodemographic characteristics and details about the participants in each study, the main results, and the assessment methods used (ordered by age and alphabetically).

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Yang et al. (2017)	106	14 years; 51% ♂ 49% ♀; normative; -	Tensor Based-Morphometry (gray matter, white matter and cerebrospinal fluid)	RPQ	Twin study (correlational study)
Schlüter et al. (2013)	22	25.2 ± 4.8; 100% ♂; normative; -	6-[18 F]-fluoro-L-DOPA (FDOPA)-positron emission tomography (PET)	PSAP	Correlation analysis (comparative study)
Dambacher et al. (2015a)	15	22.33 ± 2.35; 100% ♂; normative; -	Blood oxygenation level-dependent signal	TAP	General linear model with Fourier basis and correlations (comparative study)
Bobes et al. (2013)	54	28.93 ± 7.03 ± 0.42; 100% ♂ normative; hispanic	BOLD-contrast-weighted echoplanar (EPI) imagesROI	RPQ	Correlation analysis (comparative study)
Chester et al. (2019)	35	18.89 ± 1.11; 38% ♂ 62% ♀; normative; -	fMRIROI	ITAP	Correlation analysis (comparative study)
Emmerling et al. (2016)	15	22.33 ± 2.35; 100% ♂ normative; -	fMRI	TAP	Monte Carlo simulation iteration
Gan et al. (2015)	35	19.1 ± 0.4; 69% ♂ 31% ♀; normative; -	fMRIROI	TAP	Correlational analysis (comparative study)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Jiang et al. (2018b)	39	20.05 ± 0.92; 49% ♂ 51% ♀; normative; -	fMRIROI	TAP	Correlation analysis (comparative study)
Krämer et al. (2011)	36	24.8 ± 63.1; 100% ♂; normative; -	fMRIROI	TAP	Repeated measures ANOVA (comparative study)
Lotze et al. (2007)	16	28.6 ± 6.5; 100% ♂; normative; -	fMRIROI	TAP	Repeated measures ANOVA (comparative study)
Skibsted et al. (2017)	20	24.6 ± 2.9; 40% ♂ 60% ♀; normative; -	fMRIROI	PSAP	Regression analysis (comparative study)
Repple et al. (2018)	42	24.77 ± 2.8; 52% ♂ 48% ♀; normative; -	fMRIROI	TAP	Correlation analysis (comparative study)
Beyer et al. (2015)	40	23.3 ± 2.7; 100% ♂; normative; -	fMRIROI	TAP	Correlation analysis (comparative study)
Beyer et al. (2014)	34	22.5 years; 41% ♂ 59% ♀; normative; -	fMRIROI	TAP	Correlation analysis (comparative study)
Farah et al. (2018)	156	35.72 ± 8.61; 100% ♂ normative; 43.7% black, 32.3% caucasian	MRI scansROI	RPQ	Correlational study (comparative study)
Buades-Rotger et al. (2016)	39	23.22 ± 3.2; 100% ♀; normative; -	Resting-state fMRIROI + hormones saliva and blood (T, C and T, respectively)	STAP	Correlation analysis (comparative study)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Buades-Rotger et al., 2018 (Same data 2016)	39	23.22 ± 3.2; 100% ♀; normative; -	Resting-state fMRI ROI	STAP	Regression analysis (comparative study)
Buades-Rotger et al. (2019) (Same data 2016)	39	23.22 ± 3.2; 100% ♀; normative; -	Resting-state fMRI ROI + hormones saliva and blood (T, C and T, respectively)	STAP	Correlation analysis (comparative study)
Zhu et al. (2019a)	155	19.85 ± 1.57; 40% ♂ 60% ♀; normative; -	Resting-state fMRI ROI	RPQ	Regression analysis (comparative study)
Clinical population					
Offenders					
Chen et al. (2019)	59	17.84 ± 1.57; 100% ♂ offenders and normative; -	Stimulus-locked ERPs	RPQ and TAP	Correlation analysis (comparative study)
Hofhansel et al. (2020)	54	From 18–55; 100% ♂ violent offenders and normative; -	MRITissue Probability Maps	RPQ	Correlation analysis (Comparative study)
Kolla et al. (2021)	32	35.6 ± 9.4; 100% ♂; offenders and normative; 53% Caucasian	[11 C]CURB PET scan	RPQ	Correlational analysis (comparative study)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Pardini et al. (2014)	56	From 25.78 ± 0.96 to 29.25 ± 1.11; 100% ♂; with criminal records; 55.7% african-american	MRI scannerAmygdala volume	RPQ	Regression analysis(Longitudinal study)
Siep et al. (2019)	36	35.17 ± 7.12; 100% ♂ offenders and normative; -	Resting-state fMRIROI	RPQ	Correlation analysis (comparative study)
Zhou et al. (2006)	100	From 15–19; 100% ♂;offenders and normative; Asian	5-HT	PSAP	Correlation analysis (comparative study)
ODD, CD, ASPD and ADHD					
Aggensteiner et al. (2020)	177	From 8–18; 70% ♂ 30% ♀; ODD, CD and normative; -	fMRIROI	RPQ	Regression analysis (comparative study)
Craig et al. (2019)	233	13.0 ± 2.8; 74% ♂ 26% ♀ ODD, CD and normative; -	Glutamate concentrations in the ACC/mPFC, amygdala, insula and striatum	RPQ	Correlation analysis (comparative study)
Gan et al. (2016)	18	31.8 ± 6.5; 100% ♂ anger management problems (reactive) and normative; 50% black; 40% hispanic	fMRIROI	PSAP	t-test(comparative study)
Lozier et al. (2014)	46	From 10–17; 57% ♂ 43% ♀; CD and normative; -	fMRIROI	RPQ	Regression analysis (comparative study)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Michalska et al. (2016)	107	9–11 years old; 51% ♂ 49% ♀; CD; -	fMRI ROI	RPQ	Regression analysis (comparative study)
Naaijen et al. (2020)	254	From 8–18; 74% ♂ 26% ♀; CD, ODD and normative; -	MRI Cortical volumes and cortical thickness	RPQ	Correlation analysis (comparative study)
White et al. (2013)	59	14.90 ± 2.08; 75% ♂ 25% ♀; ODD, CD and normative; 49% african american and 51% european american	MRI caudum septum pellucidum	ABTC	One way ANOVA (comparative study)
White et al. (2016)	56	14.74 ± 2.13; 59% ♂ 41% ♀; ODD, CD and normative; -	fMRI ROI	ABTC	Regression analysis (comparative study)
Kolla et al. (2018)	40	From 18–55; 100% ♂; ASPD and normative; -	Resting-state fMRI ROI	RPQ	Correlation analysis (comparative study)
Werhahn et al. (2020)	207	13.30 ± 2.60; 72% ♂ 28% ♀; disruptive behavior + ADHD and normative; -	Resting-state fMRI ROI	RPQ	Correlation analysis (comparative study)
Non-invasive brain stimulation					
Chen (2018)	32	20.25 ± 1.34; 50% ♂ 50% ♀ normative; -	Right VLPFC tDCS	RPQ	ANOVA and correlation analysis

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
				TAP	(randomized; sham controlled)
Dambacher et al. (2015b)	32	22.14 ± 2.00; 41% ♂ 59% ♀; normative; -	Right DLPFC tDCS	RPQ TAP	Repeated measures ANOVA (pseudorandomize sham controlled)
Dambacher et al. (2015c)	64	21.89 ± 3.26; 61% ♂ 39% ♀; normative; -	Left inferior frontal cortex and the right inferior frontal cortex tDCS	RPQ TAP	One way MANOVA (pseudorandomize sham controlled)
Gilam et al. (2018)	24	26.16 ± 3.63; 40% ♂ 60% ♀; normative; -	Bilateral vmPFC tDCS	TAP	One way ANOVA(double-blind sham-controlled)
Ling et al. (2020)	92	19.98 ± 1.65; 47.8% ♂ 52.2% ♀; normative; 53.3% non-white	Bilateral PFC (HD)-tDCS	RPQ	Mann-Whitney U tests (comparative study)
Perach-Barzilay et al. (2013)	18	28.00 ± 4.68; 78% ♂ 22% ♀; normative; -	Bilateral DLPFC cTBS	SOP	Repeated measures ANOVA(randomize sham-controlled)
Riva et al. (2017)	79	21.73 ± 2.38; 52% ♂ 48% ♀; normative; -	Right VLPFC tDCS	TAP	One way ANOVA(randomize sham-controlled)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Sergiou et al. (2021)	50	37.40 ± 9.19; 100% ♂; heroin dependent; -	Bilateral vmPFC (HD)-tDCS	RPQ PSAP	Repeated measures ANOVA (double-blind, randomized, sham-controlled)
Weidler et al. (2020)	51	From 18–60; 100% ♂; AD, TU and normative; -	Right DLPFC tDCS	TAP	Multivariate ANOVA(double-blind sham-controlled)
Drug administration					
Cherek and Lane (1999)	10	27.6 ± 2.5; 100% ♂; CD; -	d,L-fenfluramine (oral)(0.2, 0.4 and 0.8 mg/kg)	PSAP	Repeated measures ANOVA(comparative study)
Cherek and Lane (2001)	10	28.1 ± 2.2; 100% ♂ CD and normative; -	D-fenfluramine (oral)(0.1, 0.2 and 0.4 mg/kg)	PSAP	Repeated measures ANOVA(comparative study)
Coccaro et al. (1996)	14	24 ± 3.55; 100% ♂ PD; -	D-fenfluramine (oral)(0.5 mg/kg)	PSAP	Correlation analysis (comparative study)
Dougherty et al. (1999)	8	30.1 ± 8.2; 100% ♂ normative; -	Acute tryptophan depletion	PSAP	Repeated measures ANOVA (comparative study)
Kötting et al. (2013)	20	11.80 ± 1.88; 50% ♂ 50% ♀; ADHD; -	Acute tryptophan depletion	PSAG	Chi-square(double-blind, randomized controlled trial)
Moeller et al. (1996)	10	25 years; 100% ♂;normative; -	Acute tryptophan depletion(25 and 100 gr)	PSAP	Repeated measures ANOVA(comparative study)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Stadler et al. (2007)	22	10.91 ± 1.77; 100% ♂ ADHD; -	Acute tryptophan depletion	Computer competitive task	Repeated measures ANOVA(double-blind,randomized controlled trial)
Zepf et al. (2008)	27	10.91 ± 1.77; 100% ♂; ADHD; -	Acute tryptophan depletion	Competitive reaction time game	Correlational analysis(double-blind within-subjectcrossover-design)
Zimmerman et al., 2012	40	30.25 ± 9.37; 100% ♂ ADHD and normative; -	Acute tryptophan depletion	PSAP	Repeated measures ANOVA (double-blind, randomized controlled trial)
Fulwiler et al. (2005)	10	29.6 ± 6.1; 100% ♂ normative; -	Citalopram (40 mg)	PSAP	Correlation analysis (comparative study)
Gowin et al. (2010)	11	28.5 ± 6.3; 64% ♂ 36% ♀; ASPD; 82% African-American	Zolmitriptan (5 mg)	PSAP	Univariate ANOVA (double-blind, placebo-controlled)
Casat et al. (1995)	6	9.0 ± 1.5; 100% ♂;ADHD; -	Methylphenidate (0.3 and 0.6 mg/kg)	PSAP	Repeated measures ANOVA (double-blind, placebo controlled)
Cherek et al. (2002b)	20	28.5 ± 1.5; 100% ♂; inmates and controls; -	Baclofen (0.07, 0.14 and 0.28 mg/kg)	PSAP	Univariate ANOVA (placebo-controlled)
Coccaro and Lee (2019)	10	33.6 ± 10.9; 50% ♂ 50% ♀; IED; -	Lorcaserim (20 mg)	TAP	T-test(double-blind placebo-controlled)

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
Cherek et al. (2002a)	12	35 years; 100% ♂; CD; -	Paroxetine (20 mg/day)	PSAP	Repeated measures ANOVA (comparative study)
Berman et al. (2009)	80	24.33 ± 7.27; 53% ♂ 47% ♀; normative; -	Paroxetine (40 mg)	TAP	Univariate ANOVA (double-blind; randomized controlled trial)
Fanning et al. (2014)	47	23.68 ± 7.81; 55% ♂ 45% ♀; normative; 68.1% caucasian	Paroxetine (40 mg)	TAP	Regression analysis (double-blind; placebo-controlled)
McCloskey et al. (2009)	56	24.23 ± 4.48; 100% ♂; normative; 78% caucasian	Paroxetine (20 mg)	TAP	Univariate ANOVA (double-blind; placebo-controlled)
Kaltsouni et al. (2021)	30	From 20–45; 100% ♀; PMDD; caucasian	Ulipristal acetate (Esmya®) 5 mg (daily/3 months)	PSAP	Correlation analysis (double-blind, randomized, placebo-controlled)
Gerra et al. (2007)	30	25 ± 5.3; 100% ♂; heroin dependent and normative; -	Buprenorphine (8.6 ± 3.5 mg daily) and methadone (80.5 ± 26.1 mg daily)	PSAP	Repeated measures ANOVA (comparative study)
Liu et al. (2017)	64	21.3 ± 1.9; 100% ♂; normative; chinese	5 mg of melatonin and 200 mg of	TAP	Univariate ANOVA (double-blind;

Authors	Sample size	Age, gender and type of group	Neuroimaging	Aggression assessment	Statistical analysis design study
Neuroimaging techniques					
Normative population					
			starch or 200 mg of starch (placebo)		randomized; controlled trial)

Note. ABTC: Aggression Behavior Teacher Checklist; ACC: Anterior Cingulate Cortex; AD: Alcohol Dependent; ADHD: Attention-Deficit/Hyperactivity Disorder; ASPD: Antisocial Personality Disorder; BOLD: Blood Oxygen Level Dependent; CD: Conduct Disorder; cTBS: Continuous Theta Burst Transcranial Magnetic Stimulation; DLPFC: Dorsolateral Prefrontal Cortex; ERP: Event-Related Potential; IED: Intermittent Explosive Disorder; ITAP: InterTemporal Aggression Paradigm; fMRI: Functional magnetic resonance imaging; mPFC: medial Prefrontal Cortex; ODD: Oppositional Defiant Disorder; PMDD: Premenstrual Dysphoric Disorder; PD: Personality Disorder; PSAG: Point Subtraction Aggression Game; PSAP: Point Subtraction Aggression Paradigm; ROI: Region of Interest; RPQ: Reactive Proactive Questionnaire; SOP: Social Orientation Paradigm; STAP: Social Threat Aggression Paradigm; TAP: Taylor Aggression Paradigm; tDCS: Transcranial Direct Current Stimulation; TU: Tobacco users; VLPFC; Ventrolateral Prefrontal Cortex; VmPFC: Ventromedial Prefrontal Cortex.

3.6.3. Reactive aggression

Initially, stimulation with transcranial direct current stimulation (tDCS) over the right VLPFC involved a reduction in reactive aggression measured with the TAP (Chen, 2018; Riva et al., 2017), but not self-reported (Chen, 2018). Furthermore, application of high definition tDCS or anodal tDCS bilaterally over vmPFC entailed reductions in reactive aggression in heroin-dependent men (Sergioui et al., 2021) and young adults of both genders (Gilam et al., 2018). In addition, inhibition of the left DLPFC through the application of continuous theta burst transcranial magnetic stimulation (cTBS) was associated with a greater increase in reactive aggression, in comparison with inhibition of the right DLPFC (Perach-Barzilay et al., 2013). Moreover, another study pointed out that only alcohol-dependent patients benefited from anodal tDCS over the right DLPFC,

given that they did not experience increases in reactive aggression after active stimulation, but they did after sham stimulation (Weidler et al., 2020).

3.6.4. Proactive aggression

Inhibition of the left DLPFC increased proactive aggression, measured with the Social Orientation Paradigm (SOP), in healthy young adults of both genders, in comparison with inhibition of the right DLPFC (Perach-Barzilay et al., 2013). This was partly supported by another study whose authors concluded that application of tDCS over the right DLPFC only reduced proactive aggression, measured with the TAP, in young men (Dambacher et al., 2015b). Moreover, a posterior study concluded that application of tDCS over the right VLPFC reduced proactive aggression (assessed with the TAP, but not self-reported) in young adults of both genders (Chen, 2018).

3.6.5. Drug administration

We also considered manuscripts that analyzed how aggression proneness was modified after drug administration. In this regard, a total of 21 studies were included, of which seven reported a direct effect on reactive aggression. Furthermore, it was necessary to include additional variables to reach significance in 12 additional studies.

It should be kept in mind that the majority of the research in this block showed robust methodological designs (i.e., double-blind, randomized, placebo-controlled...), but only a few of them considered a sample size larger than or equal to 40 participants (Berman et al., 2009, Fanning et al., 2014, Gerra et al., 2007, Kaltsouni et al., 2021, Liu et al., 2017, McCloskey et al., 2009; Zimmermann et al., 2012), which should be considered a potential risk of bias in the interpretation of the data. In any case, all the studies employed well-validated measurements of reactive aggression (e.g., PSAP, TAP, among others).

Unfortunately, only four studies mixed men and women (Berman et al., 2009, Coccaro and Lee, 2019, Fanning et al., 2014, Gowin et al., 2010), and the rest were based exclusively on men. Lastly, many of these studies did not provide information about the ethnicity of the participants. In fact, only six of them specified it (Berman et al., 2009, Fanning et al., 2014, Gowin et al., 2010, Kaltsouni et al., 2021, Liu et al., 2017, McCloskey et al., 2009).

These studies were organized according to the substance participants received. Therefore, we will initially describe the results after *d,L-fenfluramine* administration, followed by acute tryptophan depletion. Afterwards, we present results of citalopram,

zolmitriptan, methylphenidate, baclofen, lorcaserim, ulipristal acetate, buprenorphine, and melatonin.

Of the three studies that assessed *d*- or *d, L*-fenfluramine effects (enhancing 5-HT liberation and inhibiting its reuptake) on PSAP performance, only one of them obtained a direct association between *D*-fenfluramine peak levels in blood - after the administration of 0.5 mg/kg - and a reduction in reactive aggression on the PSAP in a group of young men with personality disorders (Coccaro et al., 1996). For the other two studies, it was completely necessary to consider the dose of *D*-fenfluramine administration to find a reduction in reactive aggression in young men with conduct disorders. In fact, it was necessary to administer doses of *d, L*-fenfluramine ranging from 0.4 to 0.8 mg/kg to reduce reactive aggression in boys with CD (Cherek and Lane, 1999, Cherek and Lane, 2001).

Of the six manuscripts that assessed the manipulation of tryptophan (precursor of 5-HT) levels, none of them reported a main 'drug' effect on reactive aggression measured with PSAP and PSAG. These studies were conducted in young adult males (Dougherty et al., 1999, Moeller et al., 1996) and mainly in adolescent boys with Attention-Deficit/Hyperactivity Disorder (ADHD) (Kötting et al., 2013, Stadler et al., 2007, Zepf et al., 2008; Zimmermann et al., 2012). Curiously, it was necessary to include additional variables to reach significance. In fact, significant 'drug' effects were found after considering 'hostility trait' (Dougherty et al., 1999), provocation (Kötting et al., 2013, Stadler et al., 2007), 'group' (Zimmermann et al., 2012), and 'session' (Moeller et al., 1996). Thus, Dougherty et al. (1999) concluded that only normative young men with high hostility trait reported an increase in reactive aggression after acute tryptophan depletion. Nonetheless, in adolescents of both genders with ADHD, acute tryptophan depletion increased reactive aggression after low provocation (Kötting et al., 2013; Stadler et al., 2007; Zimmermann et al., 2012). In contrast, another study reported that administration of beverages of 25 or 100 g to promote acute tryptophan depletion increased reactive aggression after 5–6 sessions/days of treatment in normative young men (Moeller et al., 1996).

After administering 40 mg of citalopram (a selective serotonin reuptake inhibitor) in healthy young men, a high response to citalopram (prolactin changes) was related to reduced reactive aggression during PSAP (Fulwiler et al., 2005).

Research assessing the role of zolmitriptan (a selective 5-hydroxytryptamine 1B/1D receptor agonist) in aggression found reduced reactive aggression after administrating 5

mg of this drug in combination with alcohol (0.4-g/kg or 0.8-g/kg) in young adults of both genders with ASPD ([Gowin et al., 2010](#)).

Regarding ADHD patients, another study reported that these boys only reported a reduction in reactive aggression (measured with PSAP) after a maximum dose of 0.6 methylphenidate mg/kg ([Casat et al., 1995](#)).

Another study assessed the effects of baclofen, a drug modulating 5-HT receptors through GABAB receptors, in young men with conduct disorder. The authors concluded that a maximum dose of 0.28 mg/kg produced a decrease in reactive aggression in these young men, but an increase in normative men ([Cherek et al., 2002b](#)).

Young adults of both genders with intermittent explosive disorder presented decreased reactive aggression after the administration of 20 mg of lorcaserin (a direct agonist at 5-HT_{2c} receptors) ([Coccaro and Lee, 2019](#)).

Four additional studies assessed whether paroxetine (a 5-HT reuptake inhibitor) decreased reactive aggression. Only one study reported a decrease in reactive aggression in a group of healthy young adult men after administering 20 mg of paroxetine ([McCloskey et al., 2009](#)). Two studies also reached significance, but it was necessary to consider the number of days administering paroxetine (specifically 16) to find significant decreases ([Cherek et al., 2002a](#)). Moreover, only young adults of both genders with a previous history of aggressive attacks showed reductions in reactive aggression after 40 mg of paroxetine ([Berman et al., 2009](#)).

A group of premenstrual women with dysphoric disorder received 5 mg of ulipristal acetate (selective progesterone receptor modulator) or a placebo daily for three months. After treatment, only the placebo group presented a negative relationship between fronto-cingulate reactivity and reactive aggression (measured with PSAP), but this association did not reach significance in the ulipristal acetate group ([Kaltsouni et al., 2021](#)).

A study assessed whether heroin-dependent patients treated with buprenorphine or methadone presented changes in PSAP in comparison with a group of normative men. Although there were no differences between the heroin patients, both groups of treated heroin-dependent patients presented higher reactive aggression than the control group ([Gerra et al., 2007](#)).

Lastly, acute administration of melatonin (5 mg) was related to higher reactive aggression (measured with the TAP) compared to the placebo in healthy young men ([Liu](#)

et al., 2017).

3.7. Psychophysiology

Of the 37 studies included in this section, 16 reported a direct association between a specific psychophysiological marker and reactive and/or proactive aggression. Moreover, 21 failed to find or did not report an association for the full sample, but of them, 13 reached significance after including additional variables (e.g., group, gender, history of victimization, among others) (Table 3).

Table 3. Main sociodemographic characteristics and details about the participants in each study, the main results, and the assessment methods used (ordered by age and alphabetically).

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	Significance
Aggression measured with self-reports						
Normative population						
Children and adolescents						
Song et al. (2020)	150	4 years; 50% ♂ 50% ♀; normative; 15% West European, 14% South Asian, 14% American	Resting SC	Little et al., (2003) self-report	Correlation analysis(comparative study)	N
Aggenstein et al. (2020)	63	From 8–18; 30% ♂ 70% ♀ ODD, CD and normative; -	SCR in response to emotional facial stimulus	RPQ	Correlation analysis(comparative study)	N
Ahmed et al. (2018)	83	From 12–18; 40% ♂ 60% ♀ normative; -	SCR in response to emotional video scenes	RPQ	Correlation analysis(comparative study)	N

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	(c) re as
Aggression measured with self-reports						
Normative population						
Children and adolescents						
Banny et al. (2014)	196	10.11 ± 0.64; 46% ♂ 54% ♀; Normative; -	Systolic and diastolic blood pressure reactivity and SCR in response to interview	CSBS	Correlation analysis(longitudinal study)	N re
Gao et al. (2018)	340	9.06 ± 0.60; 48.2% ♂ 51.8% ♀; normative; 52% African American	SC in response to video (unconditioned responses)	RPQ	Correlation analysis(comparative study)	N
Gao et al. (2015)	306	From 10–18; 45% ♂ 55% ♀;normative; 42,9% Hispanic	SC in response to laboratory (unconditioned responses and orienting responses)	RPQ	One way ANOVA(longitudinal study)	Ye
Glenn et al. (2019)	360	9.72 ± 0.62; 65% ♂ 35% ♀; normative; 76.4% African-American	Resting SCL and RSA	ABTC	Correlation analysis(longitudinal study)	N
Hubbard et al. (2002)	272	8 years; 50% ♂ 49% ♀; normative; 70% European American	HR and SCL reactivity to a computer competitive game	ABTC	Regression analysis(comparative study)	Ye
Kassing et al.	188	9–12; 65.4% ♂ 34.6% ♀; normative; 75.5%	Resting RSA and SCL	RPQ	Regression analysis(comparative	N

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	Study design
Aggression measured with self-reports						
Normative population						
Children and adolescents						
(2018)		African American			study)	
Scarpa et al. (2010)	68	9.64 ± 2.08; 62% ♂ 38% ♀;normative; 87.5% White	Resting HR, HRV and SCL	R-PRPA	Correlation analysis(comparative study)	Yes
Xu et al. (2014)	189	7.64 years; 52% ♂ 48% ♀;normative; -	Resting HR and RSA	Teachers' Ratings of Proactive and Reactive Aggression	Correlation analysis(longitudinal study)	Yes
Moore et al. (2018)	35	11.32 ± 1.07; 54% ♂ 34.6% ♀; normative; 66% European American	Resting RSA and SCL and response to aggressive task	Laboratory aggression task	Regression analysis(comparative study)	Yes
Muñoz et al. (2008)	85	15.53 ± 1.28; 100% ♂; normative; 69% African American	Resting SCL and in response to competitive task	PCS	Regression analysis(comparative study)	No
Raine et al. (2014)	334	11–17; 58.4% ♂ 41.6% ♀;normative; -	Resting HR	RPQ	Regression analysis(comparative study)	Yes
Crozier et al. (2008)	585	From 16–18; 52% ♂ 48% ♀; normative; 17% African American	Resting HR and HR reactivity to videos	RPQ	Correlation analysis(comparative study)	No re
Young adults and adults						

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	Significance
Aggression measured with self-reports						
Normative population						
Children and adolescents						
Armstrong et al. (2019)	454	20.3 ± 2.90; 33% ♂ 67% ♀ normative; 39.2% Hispanic	Resting SCR and HRR and in response to TSST	RPQ	Correlation analysis (comparative study)	Yes
Hagan et al. (2020)	159	21.6 ± 3.15; 100% ♀; normative; 32% Hispanic and 27% Pacific-Islander	PEP and RSA in response to social stress task	RPQ	Regression analysis(longitudinal study)	No
Murray-Close et al. (2017)	247	18.77 ± 0.97; 26% ♂ 74% ♀; normative; 84% Caucasian	RSA and SCL reactivity in response to social stress task	Self-Report of Aggression	Regression analysis(comparative study)	No
Murray-Close and Rellini (2012)	83	22.02 ± 2.71; 100% ♀; normative; 91% Caucasian	RSA, SBPR, DBPR, HRR and SCL in response to social competence interview	Self-Report of Aggression and Social Behavior Measure	Regression analysis(comparative study)	No
Poltavski et al. (2018)	16	20.31 ± 1.62; 69% ♂ 31% ♀;normative; -	Mean magnitude of physiological response	RPQ	Correlation analysis(comparative study)	No
Portnoy et al. (2014)	335	16.15 ± 0.89; 100% ♂; normative; 58.5% African American	Resting HR and in response to stress tasks	RPQ	Correlation analysis(comparative study)	Yes
Puhalla and	76	21.49 ± 4.14; 38% ♂ 62% ♀;normative;	HR, RSA and SCL and in response to	RPQ	Correlation analysis(comparative	Yes

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	(c) Significance
Aggression measured with self-reports						
Normative population						
Children and adolescents						
McCloskey (2020b)		51% Caucasian	TAP		study)	
Thomson and Beauchaine (2018)	104	19.85 ± 1.17; 17% ♂; 83% ♀;normative; 84% White	Resting RSA	RPQ	Correlation analysis(comparative study)	Yes
Thomson et al. (2019)	83	19.57 ± 1.10; 100% ♀; normative; 87% White	Resting RSA	RPQ	Correlation analysis(comparative study)	Yes
Wagner and Abaied (2015)	180	19.50 ± 1.04; 23% ♂; 77% ♀;normative; 94.6% White	RSA and SCL in response to social competence interview	SRASBM	Correlation analysis(comparative study)	No
Wagner and Abaied (2016)	168	19.48 ± 1.02; 23% ♂; 77% ♀;normative; 94.6% White	SCL in response to challenge task	SRASBM	Correlation analysis(comparative study)	No
Zhang and Gao (2015)	84	21.85 ± 6.67; 32% ♂; 68% ♀;normative; 34% Caucasian 20% Black	Resting RSA and in response to decision-making task	RPQ	Correlation analysis(comparative study)	Yes
Scarpa et al. (2008)	40	59.80 ± 1.81; 58% ♂; 42% ♀;normative; 88% White	Resting HR and HRV	R-PRPA	Correlation analysis(comparative study)	No

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	Significance
Aggression measured with self-reports						
Normative population						
Children and adolescents						
Clinical population						
Schoorl et al. (2016)	66	8–12; 100% ♂; ODD/CD; -	Resting HR, HRV and SCL and in response to stress (computer task)	IRPA	Correlation analysis(comparative study)	N
Brzozowski et al. (2021)	92	19.09 ± 1.25; 100% ♀;IPV perpetrators and normative; 95% White	Resting HR and resting heart rate variability expressed in standard deviation of successive interbeat intervals	RPQ	Correlation analysis(comparative study)	Yes
Zijlman et al. (2021)	127	22.42 ± 2.38; 100% ♂;Multiproblem and controls; -	Resting HR, PEP, RSA and SCL and in response to film clips	RPQ	Regression analysis(comparative study)	N
Aggression measured with laboratory tasks						
Children and adolescents						
Juujärvi et al. (2006)	48	From 8–13; 50% ♂ 50% ♀; normative; -	HR and HRV in response to PAM	PAM	Univariate ANOVA (comparative study)	N

Authors	Sample size	Age, gender and type of group	Psychophysiology	Aggression assessment	Statistical analysis; design study	Significance
Aggression measured with self-reports						
Normative population						
Children and adolescents						
Von Polier et al. (2014)	15	11.8 ± 1.9; 47% ♂ 53% ♀; ADHD; -	Acute tryptophan depletion (SCL)	PSAG	Correlation analysis(comparative study)	N
Pincham et al. (2015)	60	15.65 years; 50% ♂ 50% ♀; normative; -	ERP in response TAP	TAP	Correlation analysis(comparative study)	Ye
Young adults						
Assaad et al. (2006)	74	20.76 ± 0.72; -; normative; -	HR response to TAP	TAP	Univariate ANOVA(comparative study)	N
Hoaken et al. (2003)	64	From 20–54; 50% ♂ 50% ♀; normative; -	HR, systolic and diastolic blood preassure three moments (pre, +40 min, post-)	TAP	Regression analysis(comparative study)	Ye
Puhalla et al. (2020a)	81	22.02 ± 4.46; 32% ♂ 68% ♀; normative; 59% Caucasian	Resting HR and HRV	TAP	Correlation analysis(comparative study)	Ye

Note. ABTC: Aggression Behavior Teacher Checklist; CSBS: Children's Social Behavior Scale; HR: Heart Rate; HRR: Heart Rate Reactivity, HRV: Heart Rate Variability; IRPA: Instrument for Reactive and Proactive Aggression; PAM: Pulkkinen Aggression Machine; PCS: Peer Conflict Scale; PSAG: Point Subtraction Aggression Game; R-PRPA: Revised Parent Rating Scale for Reactive and Proactive Aggression; SC: Skin Conductance; SRASBM: Self-Report of Aggression and Social Behavior Measure.

We considered it appropriate to divide this section into two blocks. The first block is dedicated to summarizing the relationship between resting psychophysiological levels and aggression, and the second block assesses the relationship between psychophysiological markers in response to specific tasks and aggressive behavior.

3.8. Resting periods

3.8.1. Reactive aggression

Some studies showed that the resting heart rate (HR) is negatively associated with self-reported reactive aggression in children of both genders ([Raine et al., 2014](#); [Xu et al., 2014](#)) and adolescent males ([Portnoy et al., 2014](#)), although we cannot conclude that there is a homogeneous pattern of results. In fact, [Scarpa et al. \(2010\)](#) concluded that resting HR was only positively associated with self-reported reactive aggression in children of both genders. Moreover, additional research only obtained significant effects after splitting the full sample into groups (boys with and without Oppositional defiant disorder (ODD)/ conduct disorder (CD)). Thus, they concluded that resting HR and skin conductance (SC; a sympathetic marker) were positively related to self-reported reactive aggression only in boys with ODD/CD ([Schoorl et al., 2016](#)).

HR functioning is the result, at least in part, of the balance between the sympathetic and parasympathetic branches of the autonomous nervous system, and so it would be important to assess the functioning of each branch separately. For example, a parasympathetic marker, resting respiratory sinus arrhythmia (RSA), was negatively associated with reactive aggression, measured with a self-report and with a laboratory aggression task, in children who received low inconsistent discipline ([Kassing et al., 2018](#)), in adolescents ([Moore et al., 2018](#); [Xu et al., 2014](#)), and in young adults of both genders ([Moore et al., 2018](#), [Thomson and Beauchaine, 2018](#), [Thomson et al., 2019](#)). Nonetheless, not all the studies were unanimous in their conclusions. For example, [Zhang and Gao \(2015\)](#) concluded that resting RSA, Low Frequency/High Frequency-Heart Rate Variability, and the Root Mean of the Squared Successive Differences (MSSD) (other parasympathetic markers) were positively associated with self-reported reactive aggression (as well as on a laboratory task) in young adults of both genders ([Puhalla et al., 2020a](#); [Zhang et al., 2015](#)) and adults of both genders who reported high levels of witnessed community victimization ([Scarpa et al., 2008](#)). Hence, it is difficult to conclude that the low predominance of PNS during resting periods could underlie reactive aggression, except in adolescents.

3.8.2. Proactive aggression

The conclusions showed that resting HR was negatively associated with self-reported proactive aggression in children of both genders (Raine et al., 2014; Xu et al., 2014), normative young adults of both genders (Puhalla et al., 2020a), adults of both genders with high community victimization (Scarpa et al., 2008), adolescent normative men (Portnoy et al., 2014), and boys with ODD/CD (Schoorl et al., 2016).

A sympathetic marker, resting SC, was positively associated with self-reported proactive aggression in young adults of both genders (Armstrong et al., 2019) and in the presence of high inconsistent discipline in children of both genders (Kassing et al., 2018).

Regarding parasympathetic markers, resting MSSD and RSA were positively associated with self-reported proactive aggression in young adult women (Brzozowski et al., 2021; Zhang et al., 2015).

3.8.3. Psychophysiological reactivity to laboratory tasks

3.8.3.1. Reactive aggression

HR reactivity to psychosocial acute stress and on measures of reactive aggression (PAM) was negatively associated with reactive aggression in a sample of normative adolescent males (Portnoy et al., 2014) and children of both genders (Juujärvi et al., 2006), respectively. Nevertheless, HR reactivity during a computer competitive game was positively associated with self-reported reactive aggression in children of both genders (Hubbard et al., 2002). These results are congruent with another study that found an association between HR reactivity to the TAP and reactive aggression under alcohol intoxication in normative adults (Assaad et al., 2006).

Regarding systolic and diastolic blood pressure, which are regulated by carotid baroreceptors, changes to laboratory tasks were negatively associated with reactive aggression (TAP and self-reported) in normative adults and children of both genders (Banny et al., 2014, Hoaken et al., 2003).

With regard to sympathetic markers, SC reactivity during a computer competitive game was positively associated with self-reported reactive aggression in children of both genders (Hubbard et al., 2002) and in response to acute stress in young adults of both genders (Murray-Close et al., 2017, Wagner and Abaied, 2016). However, SC reactivity to facial stimuli (specifically negative faces) was negatively related with reactive aggression in children and adolescents with ODD/CD and normative children and

adolescents of both genders ([Aggensteiner et al., 2020](#)). In this regard, the interaction between this marker (SC) and RSA revealed that high RSA and low SC reactivity in response to acute stress was associated with self-reported reactive aggression in individuals who reported relational victimization ([Wagner and Abaied, 2015](#)). Additionally, in adolescents of both genders, LPP (EEG) difference scores correlated positively with high reactive aggression measured with the TAP ([Pincham et al., 2015](#)).

Regarding parasympathetic markers, RSA reactivity was negatively associated with reactive aggression (measured with a laboratory task) in adolescents of both genders ([Moore et al., 2018](#)), and positively associated, in response to a decision-making task, with self-reported reactive aggression in young adults of both genders ([Zhang et al., 2015](#)). However, high RSA reactivity to a social stress task was associated with high self-reported reactive aggression, specifically in young women who reported high levels of family conflict ([Hagan et al., 2020](#)).

3.8.4. Proactive aggression

HR reactivity to psychosocial stress and other laboratory tasks (TAP) was negatively associated with proactive aggression (measured with self-reports and TAP) in samples of normative adolescent males ([Portnoy et al., 2014](#)), young adults of both genders ([Puhalla and McCloskey, 2020b](#)), and young adult women with a previous history of sexual abuse ([Murray-Close and Rellini, 2012](#)). However, HR reactivity to ambiguous emotional videos and in response to acute stress was positively associated with self-reported proactive aggression in normative adolescent males ([Crozier et al., 2008](#)) and in boys with ODD/CD ([Schoorl et al., 2016](#)).

Regarding sympathetic markers, SC reactivity to an interview was positively associated with self-reported proactive aggression only in adolescent normative boys and those with ODD/CD ([Moore et al., 2018](#), [Schoorl et al., 2016](#)). Additionally, poor SC conditioning to fear was associated with high self-reported proactive aggression in adolescents of both genders ([Gao et al., 2015](#)). However, not all the scientific studies reinforced these results because SC reactivity to laboratory tasks (e.g., facial stimuli, psychosocial stress, competitive tasks, social competence interview) was negatively related to self-reported proactive aggression in children and adolescents with ODD/CD and normative children of both genders ([Aggensteiner et al., 2020](#)) and young adults of both genders ([Armstrong et al., 2019](#), [Moore et al., 2018](#), [Wagner and Abaied, 2016](#)).

With regard to parasympathetic markers, RSA and RMSSD reactivity in response to laboratory emotional processing tasks or acute psychosocial stress were positively

associated with proactive aggression (measured with laboratory tasks, interviews, and/or self-reported) in normative children and adolescents of both genders (Aggensteiner et al., 2020), in boys with ODD/CD (Schoorl et al., 2016), in young adults of both genders (Murray-Close et al., 2017), and in women with a previous history of sexual abuse (Murray-Close and Rellini, 2012). However, RSA in response to a challenge task was negatively associated with self-reported proactive aggression in young adults of both genders (Wagner and Abaied, 2015).

3.9. Hormones

Of the 38 articles included that assessed the hormonal correlates of the two types of aggression, only eight of them presented an initial significant association between hormones and aggression. Of the studies that initially failed to find or did not report the association, 21 of them reached significance after considering additional variables (e.g., gender, group, menstrual cycle...) (see Table 4).

Table 4. Main sociodemographic characteristics and details about the participants in each study, the main results, and the assessment methods used (ordered by age and alphabetically).

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design study
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
Armstrong et al. (2021)	552	20.34 ± 3.05; 30% ♂ 70% ♀ normative; 40% Hispanic	Two saliva samples (T and C) Baseline and after task	RPQ	Regression analysis (cross-sectional)
Chen et al. (2018b)	445	11.93 ± 0.60; 50.8% ♂ 49.2% ♀ normative; 80% African American	Single baseline saliva sample (T)	RPQ	Correlation analysis (cross-sectional)
Denson et al.	53	From 18–30; 100% ♀ normative; -	Two saliva samples (T and C)	TAP	Regression analysis (cross-sectional)

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
(2013)					sectional study)
Poustka et al. (2010)	245	15 years; 48% ♂ 52% ♀; normative; -	Single morning blood C sample	Vragenlijst Instrumentele En Reactieve Aggressie	Correlation analysis (cross-sectional study)
Clinical population					
Bakker-Huvenars et al. (2020)	114	15.4 ± 1.90; 100% ♂; ASPD, ODD/CD and normative; 81% non-European	Four baseline saliva samples (OX, T and C)	RPQ	Univariate ANOVA(longitudinal and cross-sectional study)
Lopez-Duran et al. (2009)	73	From 6–7; 55% ♂ 45% ♀; children at risk for school-age conduct problems; 91% North-Americans	17 saliva samples (C) in response to acute laboratory task	Aggression Behavior Teacher Checklist	Correlation analysis(cross-sectional study)
Stoppelbein et al. (2014)	158	9.72 ± 1.99; 100% ♀ ADHD, ODD, MDD, Tourette's syndrome or learning disorders.55% African-American	Single morning blood C sample	Aggression Behavior Teacher Checklist	Correlation analysis(cross-sectional study)
van Bokhoven et al. (2005)	172	From 12–16; 100% ♂; CD and normative; caucasian	Single saliva C sample	Aggression Behavior Teacher Checklist	Regression analysis(longitudinal study)

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
Van Bokhoven et al. (2006)	96	13, 16 and 21; 100% ♂; CD and normative; caucasian	Three annual (averaged per year) salivary T	Aggression Behavior Teacher Checklist	Three one way ANCOVAS(longitudinal study)
Aggression measured with laboratory tasks					
Böhnke et al. (2010b)	20	23 ± 2.7; 50% ♂ 50% ♀ normative; -	4 salivary samples C	TAP	Correlation analysis(cross-sectional study)
Carré and McCormick (2008)	43	21.03 ± 2.96; 100% ♂; normative; 71% caucasian	Two salivary T before and after task	PSAP	Regression analysis(cross-sectional study)
Carré et al. (2009)	99	19.51 ± 2.86; 39% ♂ 61% ♀ normative; -	Salivary T	PSAP	Correlation analysis(cross-sectional study)
Carré et al. (2010)	151	19.78 ± 1.93;; 39% ♂; normative; 84.1% caucasian	3 salivary T	PSAP	Repeated measures ANOVA(cross-sectional study)
Carré et al. (2013)	237	21.73 ± 4.66; 48% ♂ 52% ♀ normative; -	2 salivary T	PSAP	Correlation and regression analysis(cross-sectional study)

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
Carré et al. (2014)	63	26; 39% ♂; High risk and normative; 94% African American	4 salivary T	PSAP	Regression analysis(cross-sectional study)
Cote et al., 2013	49	From 18–30; 49% ♂ 51% ♀ normative; -	6 salivary T, C and estradiol	PSAP	Correlation and regression analysis(cross-sectional study)
Doughe rty et al. (1997)	12	24.8 ± 7.5; 100% ♀;normative; -	Single blood T	PSAP	Correlation analysis(cross-sectional study)
Geniole et al. (2011)	74	19.96 ± 2.43; 100% ♂;normative; caucasian	3 saliva samples response task (T and C)	PSAP	Regression analysis(cross-sectional study)
Geniole et al. (2013)	201	20 ± 2.5; 52% ♂; 48% ♀;normative; 79.3% white	Two saliva samples (pre-post task) T, C and estradiol	PSAP	Regression analysis(cross-sectional study)
MacDonald et al. (2019)	70	21 ± 3.00; 33% ♂ 67% ♀ normative; -	Five saliva samples: T, C and estradiol	PSAP	Regression analysis(cross-sectional study)
Probst et al. (2018)	40	25.91 ± 5; 100% ♀; normative; -	Salivary sample of T, estradiol, progesterone and C	UG(rejection rates)	Regression analysis(cross-sectional study)
Rinnewitz et al.	40	From 13–17; 100% ♀;	5 salivary samples T, C and HR	TAP	Correlation analysis (cross-sectional study)

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design study
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
(2019)					
Vongas and Al Hajj (2017)	72	22.01 ± 3.42; 100% ♂; normative; -	Three salivary sample of T	PSAP	Regression analysis(cross-sectional study)
Clinical population					
Gerra et al. (2001a)	40	From 20–33; 100% ♂ heroin addicts and normative; -	Five blood samples: NE, EPI, ACTH, salivary cortisol and GH and psychophysiological	PSAP	Repeated measures ANOVA(cross-sectional study)
Gerra et al. (2001b)	12	24 ± 3.55; 100% ♂ MDMA users; -	4 blood samples:ACTH, cortisol, NE andEP	PSAP	Correlation analysis(cross-sectional study)
Gerra et al. (2004)	40	From 24–35; 100% ♂ heroin addicts and normative; -	4 blood samples:EPI, ACTH, salivary cortisol and GH and psychophysiological	PSAP	Repeated measures ANOVA and correlation analysis(cross-sectional study)
Gerra et al. (2007)	30	25 ± 5.3; 100% ♂; heroin dependent and normative; -	5 blood samples:NE, EPI, ACTH and C psychophysiological	PSAP	Correlation analysis (cross-sectional study)
Experimental manipulation of hormones					
Oxytocin					
Normative population					

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design study
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
Alcorn et al. (2015)	17	32 ± 9.2; 100% ♂; normative; 53% African-American	24 IU OX	PSAP	Placebo-controlled
Campbell and Hausmann (2013)	45	From 18–21; 100% ♀; normative; -	24 IU OX	PSAP	Double-blind, placebo-controlled
Ne'eman et al. (2016)	48	27.9 ± 6.00; 58% ♂ 42% ♀ normative; -	24 IU OX	SOP	Repeated measures ANOVA
Pfundmair et al. (2018)	64	24.88 ± 6.38; 37% ♂ 63% ♀ normative; -	24 IU OX	TAP	Regression analysis (cross-sectional study) Double-blind randomized, placebo-controlled
Zhu et al. (2019b)	100	22.35 ± 2.14; 50% ♂ 50% ♀; normative; non-specified	24 IU OX	UG (rejection rates)	Repeated measures ANOVA
Clinical population					
Berends et al. (2019)	57	From 18–35; 100% ♂; CD and normative; -	32 IU OX	PSAP	Double-blind, placebo-controlled
Testosterone					

Authors	Sample size	Age, gender and type of group	Hormonal variable	Aggression assessment	Statistical analysis design
Non-experimental manipulation hormones					
Self-reported aggression					
Normative population					
Carré et al. (2017)	121	25.27 ± 4.98; 100% ♂ normative; 77.5% caucasian	Exogenous T administration (androGEL)	PSAP	T-test Double-blind placebo- controlled(cross- sectional study)
Pope et al. (2000)	23	From 20–50; 100% ♂; normative and AAs users; 60% white	Injections of T	PSAP	Double-blind; placebo- controlled RCT
Wagels et al. (2018)	103	24.17 ± 3.76; 100% ♂; normative; -	50 mg T gel	TAP	Double-blind; placebo- controlled RCT
Cortisol					
Böhnke et al. (2010a)	56	23 years; 50% ♂ 50% ♀ Normative; -	20 mg hydrocortisone + 5 saliva samples C	TAP	Repeated measures ANOVA (cross- sectional study)
Gowin et al. (2013)	67	31.5 ± 9.6; ♂? ♂? ♀? ♀; inmates, SUD; -	20 mg C	PSAP	Double-blind, placebo- controlled

Note. AAs: Anabolic-Androgenic Steroids; ABTC: Aggression Behavior Teacher Checklist; ACTH: Adrenocorticotrophic Hormone; ADHD: Attention Deficit Hyperactivity Disorder; ASPD: Antisocial Personality Disorder; C: Cortisol; CD: Conduct Disorder; EPI: Epinephrine; GH: Growth Hormone; IU: International Units; MDD: Major Depressive Disorder; MDMA: Methylenedioxymethamphetamine; NE: Norepinephrine; ODD: Oppositional Defiant Disorder; Ox: Oxytocin; PSAP: Point Subtraction Aggression Paradigm; RCT: Randomized Controlled Trial; RPQ: Reactive Proactive Questionnaire; SOP: Social Orientation Paradigm; SUD: Substance Use Disorder; TAP: Taylor Aggression Paradigm; T: Testosterone; UG: Ultimatum Game.

Based on the characteristics of the studies included, we decided to divide this section into three parts. The first part is dedicated to baseline hormonal levels, and the second part is dedicated to analyzing how changes in hormonal levels (endogenous levels without external manipulation) in response to laboratory tasks are related to changes in proneness to aggression. Then, the third block is dedicated to summarizing how hormonal manipulation (exogenous administration) affects aggressive behavior.

3.9.1. Baseline hormonal levels

3.9.1.1. Reactive aggression

Focusing on reactive aggression, whereas cortisol (C) levels were negatively associated with self-reported reactive aggression in a study that only included girls with psychiatric disorders such as ADHD, ODD and/or Tourette's syndrome ([Stoppelbein et al., 2014](#)), in men the results were mixed. In fact, salivary baseline C levels were only positively associated with self-reported reactive aggression in normative boys and boys with CD ([Van Bokhoven et al., 2005](#)), whereas another study concluded that C (measured in blood) was negatively related to this kind of aggression in normative adolescent men ([Poustka et al., 2010](#)).

Baseline testosterone (T) in blood levels was negatively associated with reactive aggression measured with the STAP in young normative women ([Buades-Rotger et al., 2016](#)). In contrast, additional research concluded that high basal T predicted reactive aggression (measured with the TAP) when C basal levels were high in young normative women ([Denson et al., 2013](#)). In boys (normative and with CD), high baseline T levels were also related to high self-reported reactive aggression ([Van Bokhoven et al., 2006](#)). Finally, women with lower baseline estradiol who scored high on self-centered impulsivity presented higher reactive aggression (measured with the PSAP) than those with higher baseline estradiol and low self-centered impulsivity scores ([Geniole et al., 2013](#)).

3.9.2. Proactive aggression

In the case of proactive aggression, as in reactive aggression, C was negatively associated with self-reported proactive aggression in girls with psychiatric disorders such as ADHDM, ODD, or Tourette's syndrome ([Stoppelbein et al., 2014](#)), normative young adult women ([Armstrong et al., 2021](#)), and adolescent men ([Poustka et al., 2010](#)). Furthermore, self-reported proactive aggression was positively related to baseline T levels in normative adolescent boys with CD ([Van Bokhoven et al., 2006](#)) (see [Table 4](#)).

3.9.3. Hormonal changes in response to a laboratory task

Of all the studies included in this section, only two found significant associations with reactive aggression. Both were conducted with healthy young adult men ([Carré and McCormick, 2008](#), [Geniole et al., 2011](#)). One of them reported that aggression (measured with the PSAP) was positively associated with changes in salivary T ([Carré and McCormick, 2008](#)), and the other reported that T concentrations 10-min after initiating the laboratory task were positively correlated with reactive aggression ([Carré et al., 2013](#), [Carré et al., 2014](#); [Cote et al., 2013](#); [Geniole et al., 2011](#)).

Of the research that did not find an initial significant association or did not conduct analyses of the full sample, six reached significance after considering additional variables along with hormonal values. Specifically, changes in T concentrations were positively related to reactive aggression on the PSAP only in young adult men, specifically those in the losing condition ([Carré et al., 2009](#)) and after being provoked without reward ([Carré et al., 2010](#)). In young adult women, the results were affected by the menstrual cycle. In fact, plasma T levels correlated negatively with reactive aggression only during the midfollicular phase ([Dougherty et al., 1997](#)). Nonetheless, an additional study concluded that, in healthy young women, baseline T levels were positively correlated with reactive aggression in a laboratory task (Ultimatum Game), with this association being stronger as C levels rose ([Probst et al., 2018](#)).

Finally, four other studies conducted only in men reported that C and adrenocorticotrophic hormone changes in response to laboratory tasks only correlated positively with reactive aggression (measured with the PSAP) in healthy and normative young men, but not in heroin addicts or patients receiving methadone. However, noradrenaline and epinephrine in response to laboratory tasks were positively correlated with reactive aggression in normative men and in men with a heroin addiction or receiving methadone ([Gerra et al., 2001a](#), [Gerra et al., 2001b](#), [Gerra et al., 2004](#), [Gerra et al., 2007](#)).

3.9.4. Experimental manipulation of hormonal levels

Research was also conducted that manipulated the hormonal levels to assess how they, in turn, affected aggression proneness (measured with laboratory tasks such as PSAP, TAP, ultimatum game, and the social orientation paradigm). Most of them assessed oxytocin (OX) manipulation, but others also included T and C.

Two of the six studies concluded that manipulating OX levels involved reductions in reactive aggression in healthy young adults of both genders ([Berends et al., 2019](#),

Ne'eman et al., 2016), but the rest of the studies failed to report initial significant effects (Alcorn et al., 2015, Campbell and Hausmann, 2013, Pfundmair et al., 2018, Zhu et al., 2019b). Of these studies, three reached significance after considering gender (Zhu et al., 2019b) and trait anxiety (Campbell and Hausmann, 2013, Pfundmair et al., 2018). Thus, OX administration reduced reactive aggression in healthy young men, but not in women (Zhu et al., 2019b). Another study concluded that women with high anxiety presented higher reactive aggression during the PSAP than women with low anxiety levels, but the differences disappeared in those who received OX (Campbell and Hausmann, 2013). Contrary to previous results, the other study concluded that, only after provocation, young adults of both genders with low trait anxiety who received OX and were rejected presented higher reactive aggression than those who received the placebo (Pfundmair et al., 2018).

With regard to T and C, Pope et al. (2000) found a direct association with variations in reactive aggression during laboratory tasks, but the rest of the studies did not (Böhnke et al., 2010a, Carré et al., 2017, Gowin et al., 2013). Specifically, administration of T increased reactive aggression in androgenic-anabolic steroid users (Pope et al., 2000). After including dominance along with T administration (Carré et al., 2017) and the number of sections in the experimental procedure with C manipulation (Böhnke et al., 2010a), two studies reached significance. In the first case, Carré et al. (2017) concluded that T increased reactive aggression in healthy young men who scored low on trait dominance. Moreover, Böhnke et al. (2010a) pointed out that C increases were related to higher reactive aggression in healthy young adults of both genders in a block of several trials, in comparison with the placebo group.

4. Discussion

The results described in our review allow us to conclude that genetic factors contribute approximately 45% of the explained variance in reactive and proactive aggression, with 60% of these factors shared by both types, especially, for overt and physical expression forms. In fact, only a small percentage of genes (less than 10%) are specific to each type of aggression, although heritability seems to be more important in men than in women. It is also important to highlight that the two types of aggression presented opposite allelic risk associations with neurotransmission systems. Specifically, low functioning variants of the MAOA-LPR genotype dysregulated serotonergic and monoaminergic functioning, increasing the risk of reactive aggression, whereas high functioning variants facilitated proactive aggression. Other non-shared variants also affected serotonergic, vasopressin and dopamine neurotransmission or other basic cell

processes. Brain structural analysis only revealed an overlapping between the two types of aggression and alterations in the volume of the amygdala and temporal cortex. Moreover, during resting periods, high medial PFC activation equally facilitates proneness to both types of aggression. Specifically, whereas stimulation with non-invasive brain techniques over the right VLPFC and DLPFC reduced proneness to both types of aggression, inhibition of DLPFC facilitated their expression. Finally, psychophysiological, and hormonal correlates, in general, did not make it possible to differentiate between the two types, given that they were equally related to each type (See [Supplementary Table 2](#)). Initially, the main objective of our study was two-fold. The first aim was to summarize the biological correlates underlying reactive and proactive aggression. The second was to use neuroscientific data to help to determine whether the two types of aggression should be considered mutually exclusive concepts or complementary. Most of the scientific research included in this review presented correlational and cross-sectional research, and only a few studies showed robust experimental designs (e.g., randomized controlled trials, double-blind, placebo-controlled, etc.). Even though clinical and violent populations were included, most of the data were obtained in normative young adults of both genders. The review also highlighted that some studies only provide results for reactive aggression, which could affect the interpretation of the data (e.g., drug administration, hormonal response to laboratory tasks, and hormonal manipulation), specifically the analysis of proactive aggression.

Regarding the reported effect sizes of the studies included in this review, the majority calculated group differences by including partial eta squared as a measurement of effect size in the ANOVAs ([Richardson, 2011](#)). Many of them presented effect sizes ranging from small ($\eta^2 = 0.01$) to moderate ($\eta^2 = 0.06$). [Riva et al. \(2017\)](#) and [Carré et al. \(2013\)](#) employed Cohen's d for calculating t-test effect sizes. In both cases, Cohen's d presented a value lower than .500. Thus, we can conclude that those group differences were small ([Cohen, 1992](#)). This was similar for those studies employing correlational and regression analysis. The correlational coefficient (r) and standardized regression (beta or b) coefficients were used to measure the strength of the associations ([Peterson and Brown, 2005](#)). Most of these associations included in our review ranged from small to moderate (r and b standardized coefficient lower than .400). Among those studies reporting high coefficient correlation or standardized beta coefficient, most of them presented small sample sizes and obtained significant effects after subdividing the full sample into subgroups ([Carré and McCormick, 2008](#), [Carré et al., 2009](#); [Cote et al., 2013](#); [Dougherty et al., 1997](#); [Fulwiler et al., 2005](#); [Jiang et al., 2018b](#); [Kolla et al., 2018](#); [Repple et al., 2018](#); [Scarpa et al., 2008](#)).

Conclusions derived from twin studies allow us to conclude that heritability (genetic factors) accounted for approximately 45% of the explained variance in both types of aggression, with the rest mainly explained by non-shared environmental factors. Approximately 60% of the genetic factors were shared by both types, especially for overt and physical expression forms, and less than 11% of these factors were specific to each type of aggression (Brendgen et al., 2006, Paquin et al., 2014, Tuvblad et al., 2009). Based on these data, we can conclude that a common genetic etiology for both types is modulated by experience (non-shared environmental factors), which conditions the predominance of one type over the other. These results apparently contradict the existence of a dichotomous model of mutually exclusive concepts, and they support a complementary vision of the two types of aggression or even the distribution of both types of aggression in a continuum. In any case, several authors speculated that this high correlation between the two forms of aggression could be explained, at least in part, by the fact that the self-reports employed to measure aggression exclusively consider overt and physical forms of aggression (Brendgen et al., 2006, Paquin et al., 2017). Hence, sharing a considerable percentage of genetic factors might be exclusive to overt and physical expression forms of both types of aggression. In specific contexts both forms might complement each other. Maybe an initial offensive/defensive physical attack (short-term) could also have preventive effects (long-term). Conversely, introducing other factors in heritability or genetic studies such as subtle or passive forms of anger expression, motivations for expression, feelings underlying perpetration or factors prone to expression, personality traits, among others, might reduce the percentage of genetic factors shared between both. Moreover, heritability seemed to vary considerably depending on gender, and Baker et al. (2008) indicated that heritability was extremely important in men, whereas in women both forms of aggression were mainly explained by non-shared environmental factors. Hence, the importance of gender in the interpretation of the rest of the biological markers summarized in this review should be kept in mind.

The analysis of allelic risk factors for aggression proneness revealed that MAOA gene was equally involved in both forms of aggression, but the allelic risk factors differed, presenting opposite effects. Whereas low functioning allele variants such as MAOA promoter region (MAOA-LPR) alleles facilitate reactive aggression, high functioning variants were associated with proactive aggression in normative and offenders (Gallardo-Pujol et al., 2013, Hemmings et al., 2018, Kolla et al., 2014, Kuepper et al., 2013, Schlüter et al., 2016, Van Donkelaar et al., 2020, Zhang et al., 2016). Low functioning variants (or those with low transcription efficiency) were related to worse serotonergic and catecholaminergic (i.e., dopamine, norepinephrine, and epinephrine)

neurotransmission regulation through their effects on the catalyzation of the oxidative deamination, respectively. Their effects on neurotransmission should not be understood as a generalized decrease or increase in 5-HT or monoaminergic total levels throughout the central nervous system, given that richness of MAO activity varies in different brain structures (Fowler et al., 1987). Hence, it would be better to conceive this as an inefficient neurotransmitters system for dealing with situations entailing high levels of uncertain or negative events (Caspi et al., 2003, Caspi et al., 2010). A better understanding of their violence facilitation role could be achieved by assessing the link between these low transcription variants with alterations in specific brain networks. In this sense, carriers of low expression MAO-LPR variants tend to respond with a limbic hyperreactivity to processing emotional stimuli, interpreting certain stimuli as provoking and, in turn, facilitating reactive aggressive responses (Lee and Ham, 2008, Meyer-Lindenberg et al., 2006). Neuroimaging studies also showed that monoaminergic system alterations, such as dopaminergic and serotonergic alterations, facilitated proneness to reactive aggression. Specifically, dysregulations in dopaminergic (a neurotransmitter metabolized by MAOA) functioning in the midbrain, caudate nucleus, and putamen in young adult men explained higher reactive aggression (Schlüter et al., 2013). Some of those brain structures belong to the nigrostriatal dopaminergic pathway, which has been classically involved in voluntary movement, but their role for reward prediction errors and interfering with salience of environmental stimuli has also been suggested (Bissonette and Roesch, 2016, Horvitz, 2000). Therefore, dopaminergic degradation alterations in this pathway might affect the detection of potential threatening or other relevant signals, facilitating in turn, proneness to aggression.

It should be kept in mind that other allelic risk variants were found that were not necessarily shared by both types of aggression. Reactive aggression was also associated with less efficient 5-HT transportation to recycle into the neurotransmitter pool (STin2 VNTR12 of the SLC6A4 gene) in offender men and normative women (Hemmings et al., 2018, Peeters et al., 2020), metabolism of catecholaminergic neurotransmission (carriers of Met alleles of COMT gene) (Zhang et al., 2016) and alterations in AVP 1B receptors (Luppino et al., 2014), both of which are involved in deficits in emotional processing and/or regulation. Other allelic variants were found that affected the cell cycle and transcription during cell division (Van Donkelaar et al., 2020), which might be important in explaining morphological brain alterations in individuals with a certain proneness to reactive aggression. Emotional processing biases along with mood alterations explained by serotonergic dysregulation might facilitate reactive aggression. In fact, this mood lability was sustained by low activity STin2 VNTR polymorphism, which has usually been associated with mood dysregulations and/or disorders (Anguelova et al., 2003).

Nonetheless, the high functioning variants of these genotypes seemed to be associated with low emotionality aggression, that is, proactive. In fact, to develop this kind of aggression, usually known as ‘cold-blooded’, it is necessary to have good control over one’s emotions in order to anticipate how current reactions might affect the near future. However, this did not involve better emotional abilities, as we discuss below.

Proactive aggression was associated with low functional alleles of the DRD4 gene (Cherepkova et al., 2015) and allelic variants affecting transcriptional regulation of OXTR (Fragkaki et al., 2019), both of which were related to alterations in social cognition. These might affect social cognition through its interfering effects on the salience of social relevant and emotional stimulus, establishment of satisfactory social interactions or social rewarding processes, among others (Crespi, 2016). Additionally, SNP were also found that affected morphological cell processes, such as 1062535 and rs13381941 (Lazebny et al., 2020). This might explain certain brain structural alterations or vulnerability to developing these alterations when these individuals are exposed to social risk factors. For example, it might explain the association between alterations in liberation of glutamate in striatal, which entailed high self-reported proactive aggression in normative and CD adolescents of both genders (Siep et al., 2019).

As can be seen above, reactive aggression has mainly been associated with genetic alterations in the transportation and degradation of serotonin. Therefore, it seems logical that interventions focused on these neurotransmitter pathways might reduce aggression proneness, especially in aggressive individuals such as children with CD, ODD, or ADHD, in adults with antisocial personality disorder or intermittent explosive disorder, and/or normative individuals. In fact, several experimental studies assessed how administering specific drugs to regulate directly serotonin neurotransmission reduced reactive aggression (Berman et al., 2009, Cherek and Lane, 1999, Cherek and Lane, 2001; 2002b; Cherek et al., 2002b; Coccaro et al., 1996; Coccaro and Lee, 2019; Fulwiler et al., 2005; Gowin et al., 2010; McCloskey et al., 2009), whereas decreases in serotonin levels through tryptophan depletion treatment increased it in normative adolescents as well as in adolescents with ADHD (Dougherty et al., 1999, Kötting et al., 2013, Moeller et al., 1996, Stadler et al., 2007, Zepf et al., 2008; Zimmermann et al., 2012). However, previously described studies employed drugs affecting serotonergic pathways at different levels (e.g., inhibiting neurotransmitter reuptake, favoring postsynaptic receptors activity, favoring neurotransmitter liberation, etc.). Thus, we cannot conclude that a generalized malfunction of serotonergic neurotransmission exists, nor can we assume a generalized increase or decrease of 5-HT, given that not only the mentioned drugs favor liberation of this neurotransmitter. However, we

hypothesized that punctual reductions of reactive aggression in laboratory tasks after improvements in regulation of serotonin neurotransmission, especially inhibiting 5-HT reuptake, might be explained by the initial tendency of this kind of drugs to reduce anxiety, which in turn increases the threshold for reacting impulsively (Romero-Martínez et al., 2019). That is, regulating mood lability specifically reduces anxiety levels, which might contribute to reducing the risk of reacting aggressively as impulsivity declines.

The administration of other drugs that affect 5-HT receptors indirectly through GABAB receptors (baclofen) also reduced reactive aggression in inmates (Cherek et al., 2002b). In the same way, there were also other neurotransmitters and hormones involved that did not directly affect serotonergic pathways but reduced reactive aggression. Concretely, the administration of melatonin (hormone) in normative adults (Liu et al., 2017), buprenorphine or methadone (opioids) in heroin dependent patients (Gerra et al., 2007) and ulipristal acetate (selective progesterone receptor modulator) in women with premenstrual dysphoric disorder. This highlights how we cannot conclude that reactive aggression was only explained by a general disruption of 5-HT neurotransmission. Moreover, some of these substances, such as d,L-fenfluramine, applied in previous research, not only favor liberation of 5-HT, but affect liberation of other neurotransmitters such as dopamine and noradrenaline (Rothman et al., 2003). In this sense, dopamine proved to be crucial for reward brain circuits (Horvitz, 2000), which would affect aggressive behavior proneness interfering with future reinforcement or not of this kind of aggressive behavior. This would also be supported by research which has registered decreases in reactive aggression in patients with ADHD receiving methylphenidate (Casat et al., 1995). These patients tend to experience a regulation of dopaminergic and noradrenergic regulation after sustained treatment with this drug (Engert and Pruessner, 2008). Whether we attend to those substances regulating dopaminergic pathways, it seemed logical that favoring their functioning might reduce the establishment of rewarding benefits after perpetrating reactive aggression. Thus, we cannot diminish the role of other neurotransmitters, only paying attention to 5-HT, without considering additional neurotransmitters and hormones. Unfortunately, there is a gap in the scientific literature about whether certain drugs might interfere with proactive aggression. To answer this question, it is necessary to discuss the results of non-invasive brain stimulation techniques to treat proactive aggression to clarify the contribution of certain brain circuits to each type of aggression.

All the non-invasive brain stimulation techniques were applied on different regions of the PFC in normative young adults of both genders. The pattern of association was

similar for both kinds of aggression; in other words, their proneness was facilitated by stimulation of the high medial PFC and inhibition of the left DLPFC (Perach-Barzilay et al., 2013). Conversely, stimulation of the right VLPFC (Chen, 2018; Riva et al., 2017) and DLPFC (Dambacher et al., 2015b, Weidler et al., 2020) reduced their expression. However, one result only showed interference with reactive aggression. Specifically, bilateral stimulation of the vmPFC only reduced its proneness in normative (Sergiou et al., 2021) and clinical populations (Gilam et al., 2018). All these studies measured aggression with laboratory tasks; curiously, those that also included self-reports to measure aggression failed to report significant changes (Chen, 2018, Ling et al., 2020; Sergiou et al., 2021). Thus, it should be concluded that these techniques might interfere with current aggression reactions, but they do not modify the general tendency to manifest aggression reactively or proactively.

Neuroimaging studies also paid attention to the existence of morphological brain differences that explained aggression differences. In this case, both types of aggression shared morphological alterations in the amygdala (Bobes et al., 2013, Farah et al., 2018, Naaijen et al., 2020, Pardini et al., 2014, Yang et al., 2017, Zhu et al., 2019a) and temporal cortex (Yang et al., 2017, Zhu et al., 2019a). Conclusions about enlargement or shortening of the GVM of the amygdala, especially right-sided, were mixed. Nonetheless, reactive aggression involved shortening of the medial temporal cortex and proactive aggression involved its enlargement. However, the contribution of these structures to emotion processing was clear, especially in detecting the emotional valence (e.g., facial, prosody, etc.).

There was no other overlapping between morphological volume associations and aggressive behavior proneness. Specifically, reactive aggression was related to increases in the left putamen volume and decreases in the right middle and superior temporal gyrus and insula (Hofhansel et al., 2020, Naaijen et al., 2020, Yang et al., 2017, Zhu et al., 2019a). Curiously, these structures are involved in processes such as emotion driven decisions, discriminating emotional prosody, and/or facial emotion processing (Eimontaite et al., 2019, Uddin et al., 2017, Zhang et al., 2018). Therefore, these brain structures are critical for processing contextual stimuli, which in turn lowers the threshold for reacting impulsively with aggression after misunderstanding contextual stimuli. Moreover, these misinterpretations frequently affect inner emotional states, which also facilitate aggression proneness.

Proactive aggression is related to decreases in the volume of the PCC and lateral frontal cortex, but with a larger volume of the DLPFC (bilaterally), medial frontal cortex, ACC

(bilaterally), inferior and medial temporal cortex in both genders (Bobes et al., 2013, Farah et al., 2018, Naaijen et al., 2020, Pardini et al., 2014, Yang et al., 2017, Zhu et al., 2019a). These brain structures are involved in elaborating plans, intervening in decision-making processes, and regulating attentional focus, among others (Brown and Braver, 2005, Leech and Sharp, 2014, Stevens et al., 2011).

We also found studies that detected a certain degree of overlapping between the two types of aggression and brain functioning. Specifically, high medial PFC activation during resting periods meant high proneness to both types of aggression (Siep et al., 2019). Another study provided evidence that the N2 component of event-related potentials (in PFC) equally facilitates proneness to reactive and proactive aggression (Chen et al., 2019). This ERP component might be related to cognitive control before responding to confrontation. Therefore, both types of aggression might be initiated by the same structure, but the rest of the processes were supported by other brain structures, which explains the differences in expression. In fact, it would be necessary to understand the functional brain connectivity underlying each type.

It is important to keep in mind that numerous studies assessed reactive aggression in normative and clinical populations of both genders, but proactive aggression was mainly measured in men. Thus, this made it difficult to draw conclusions because gender was extremely important in understanding these results. For example, reactive aggression involved high activation of the PFC, ACC, striatum, amygdala, ventral striatum, and insula in both genders (Gan et al., 2015; Skibsted et al., 2017). Furthermore, the higher the reactive aggression proneness (men with serious difficulties controlling anger expression), the higher the activation of the insula, putamen, left inferior frontal operculum, and lower vmPFC and precuneus (Gan et al., 2016). Nonetheless, after splitting the genders, the patterns of activation differed sufficiently to explain reactive proneness in men and women (Repple et al., 2018).

In men, reactive aggression proneness involved high activation of the dorsal medial PFC, right inferior orbital gyrus/ACC/right rectal gyrus, insula, left frontal lobe, left inferior parietal lobe, bilateral putamen/globus pallidus, thalamus, caudate, and left hemisphere of the cerebellum (Dambacher et al., 2015a, Emmerling et al., 2016, Lotze et al., 2007, Repple et al., 2018). Proactive proneness involved high activation of the right parietal lobe and the right superior temporal gyrus (Emmerling et al., 2016).

Nevertheless, it is possible to mention that functional connectivity was studied in samples composed of young adults of both genders together. Specifically, reactive aggression entailed enhanced connectivity between the PCC and the parahippocampus,

the left amygdala, and the precuneus, diminished functional connectivity between the left omPFC and the right angular gyrus and bilateral caudate nucleus, and reduced functional connectivity between the right omPFC and the right caudate nucleus and between the vmPFC and the frontoparietal network (dorsolateral PFC, superior parietal lobule) and the cingulo-opercular network (dorsal anterior cingulate cortex) and between the right anterior insula and the nucleus caudate ([Chester et al., 2019](#), [Jiang et al., 2018b](#), [Werhahn et al., 2020](#)). One could speculate about the importance of these brain networks for behavioral control and impulsivity, given that high levels of impulsive aggression in ADHD patients have been linked with PFC and cingulate cortex morphological alterations, whereas deficits in inhibitory control of these patients have been associated with the hypoactivity of the lateral PFC with the insula and the amygdala ([Puiu et al., 2018](#)). In the case of proactive aggression, it involved reduced functional connectivity between the left PCC and the right DLPFC, right IPL, right mPFC/ACC, and bilateral precuneus, as well as reduced connectivity between the dorsal striatum and the insula and supplemental motor area and high functional connectivity between the left DLPFC and the left inferior parietal lobes ([Chester et al., 2019](#), [Werhahn et al., 2020](#), [Zhu et al., 2019a](#)).

Reactive aggression, or ‘hot-blooded’ aggression, would be expected to entail basal or resting psychophysiological hyperreactivity; in contrast, hypoarousal would be associated with instrumental and/or proactive aggression. Nevertheless, results of this review were far from homogeneous, specifically for reactive aggression. Most of the studies pointed in the following direction. That is, proactive aggression involved low resting HR, but high sympathetic (e.g., SC levels) and parasympathetic activation (e.g., RSA, LF/HF, and MSSD) ([Armstrong et al., 2019](#), [Brzozowski et al., 2021](#), [Kassing et al., 2018](#), [Portnoy et al., 2014](#), [Puhalla et al., 2020a](#), [Schoorl et al., 2016](#); [Zhang et al., 2015](#)). Some studies pointed in the same direction for reactive aggression ([Raine et al., 2014](#), [Portnoy et al., 2014](#), [Puhalla et al., 2020a](#), [Scarpa et al., 2008](#), [Schoorl et al., 2016](#); [Xu et al., 2014](#); [Zhang et al., 2015](#)), but others found the opposite pattern of results for HR ([Scarpa et al., 2010](#), [Schoorl et al., 2016](#)) and RSA ([Moore et al., 2018](#), [Kassing et al., 2018](#), [Thomson and Beauchaine, 2018](#); [Xu et al., 2014](#)). Apparently, it was difficult to conclude that there was simultaneous high activation of sympathetic and parasympathetic branches of the ANS. However, this can be explained from a methodological point of view, especially applying statistical interpretation, given that all the studies included were correlational. Hence, the results should be understood as a tendency in relationships between variables, and not as mutually exclusive results (e.g., the same individuals experiencing simultaneous activation of both systems). Thus, it is difficult to conclude that there is a differential resting pattern of ANS functioning for each type of

aggression. In any case, we argue against the clinical value of a resting period because the reliability of these data tends to be low and dependent on multiple factors.

Results for the psychophysiological response to laboratory tasks also offered mixed conclusions and do not allow us to differentiate between the two types of aggression even though we did not consider the laboratory task employed for promoting psychophysical changes. However, if we restrict the interpretation to studies that employed laboratory tasks assessing aggression in normative individuals of both genders, we can conclude that reactive aggression involved higher HR and sympathetic reactivity along with low parasympathetic changes in children and adults ([Assaad et al., 2006](#), [Hubbard et al., 2002](#), [Moore et al., 2018](#)). Based on this, we can draw conclusions about sympathetic hyperreactivity and a low vagal tone. However, it should be kept in mind that [Juujärvi et al. \(2006\)](#) concluded just the opposite, that is, reduced HR reactivity in normative children of both genders was related to higher reactive aggression. Regarding proactive aggression, participants presented a different pattern, that is, a general hypoarousal (reduced HR, SC, and RSA reactivity) ([Puhalla and McCloskey, 2020b](#), [Moore et al., 2018](#), [Wagner and Abaied, 2015](#)), with both sharing reduced parasympathetic functioning. Hence, the reduced or low parasympathetic activation of the reactive aggression corresponds to polyvagal theory, given that this model stated that aggressive behavior proneness was explained by poor functioning of the parasympathetic branch of the ANS (reduced vagal tone) in maintaining the balance with the sympathetic branch ([Porges, 2011](#)). As proactive aggression tends to be driven by low emotionality, it seemed logical to conclude that this kind of aggression did not entail significant changes in ANS markers. That is, a generalized hypoarousal underlies the aggression without provocation.

Focusing on the psychophysiological response to emotional stimuli in normative and clinical populations of both genders allows us to conclude that low SC reactivity or changes in response to these tasks involved high reactive and proactive aggression ([Aggensteiner et al., 2020](#), [Armstrong et al., 2019](#), [Wagner and Abaied, 2016](#)). However, it would be necessary to explore whether low changes implied a poor response or, conversely, the maintenance of heightened levels of sympathetic activation throughout this task. However, this low arousal changes could also be attributed, in general, to a reduced ability to deal with emotions (e.g., high alexithymia, low empathic abilities...) which facilitate aggression proneness (both types). The absence of a specific psychophysiological pattern of response to each type of aggression was similar for the response to acute laboratory stressors (e.g., social competence interview, preparing a speech about their faults, decision-making tasks, etc.). Specifically, both types of

aggression involved low HR reactivity ([Murray-Close and Rellini, 2012](#), [Portnoy et al., 2014](#)) and high SC ([Murray-Close et al., 2017](#), [Schoorl et al., 2016](#), [Wagner and Abaied, 2016](#)). Nonetheless, reactive aggression entailed high RSA ([Hagan et al., 2020](#), [Wagner and Abaied, 2016](#); [Zhang et al., 2015](#)). Thus, the interpretation of further studies should consider whether the polyvagal theory should be modified/accommodated to understand psychophysiological functioning and its association with aggressive behavior when coping with different situations. Based on the results, we cannot conclude that a specific pattern exists that can be attributed to each type of aggression without considering contextual factors.

Lastly, hormonal correlates of aggressive behavior offered little information to categorize aggressive behavior and differentiate between the two types. Only basal C levels were associated with aggression in normative and clinical populations of both genders, with the rest of the studies failing to assess OX basal levels. In this regard, reactive aggression had a mixed positive ([Van Bokhoven et al., 2005](#)) and negative association with basal C levels ([Poustka et al., 2010](#), [Stoppelbein et al., 2014](#)), but proactive aggression was only negatively associated with this hormone ([Armstrong et al., 2021](#), [Poustka et al., 2010](#), [Stoppelbein et al., 2014](#)). The differences between both types of aggression could be attributed to participants' perception of whether the laboratory task is a potential threat or not and the subsequent emotional cascade. Individuals perceiving an imminent threat who attack immediately would experience an increase of negative mood and rising C levels (reactive aggression), but those who perceive the situation with low emotionality and delay their attack might experience a decrease in C levels (proactive aggression). Hence, it depends on their perception of a potential threat and underlying/associated mood. However, for understanding the association between C baseline levels with aggression it would be important to consider the potential interaction with other hormones such as T, but also participants gender.

Regarding baseline T levels, gender seemed to be relevant because the levels were negatively associated with reactive aggression in women ([Buades-Rotger et al., 2016](#)), but positively in men ([Van Bokhoven et al., 2006](#)). Additionally, reactive and proactive aggression were positively associated with T in clinical and normative men ([Van Bokhoven et al., 2006](#)). High baseline T levels could be attributed to the assessment of potential threats, which conditions subsequent behavior. In the case of women, it should be necessary to control the potential role of menstrual cycles to understand the association with aggression. Anyway, as we discussed above; basal levels tend to offer little information about the inner functioning. Moreover, we also argue against the usefulness of resting or basal levels as reliable correlates of reactive or proactive

aggression. In fact, these variables offered confounding conclusions. Hence, these data should be complemented by dynamic functioning (e.g., in response to tasks) of endocrine glands through changes in secretory levels. Unfortunately, studies assessing this focused exclusively on reactive aggression.

T levels have frequently been found to be a correlate of proneness to aggressive behavior. As expected, [Carré and McCormick \(2008\)](#) and [Geniole et al. \(2011\)](#) found that this was important in men, but not in women ([Carré et al., 2009](#), [Carré et al., 2010](#)). A previous study offered some background to understand the existence of gender differences. In fact, after considering the menstrual cycle in women, this factor modulated the association between T and reactive proneness ([Dougherty et al., 1997](#)). Moreover, a later study also found a positive association between T and reactive aggression after considering the role of C in normative young-adult women (Probst et al., 2017). Some research pointed out that T was closely associated with aggression when C levels were low, due to the negative feedback between the systems that regulate their liberation at hypothalamic level ([Dekkers et al., 2019](#)), but most of this research only includes baseline levels for calculating the imbalance between both hormones ([Sollberger and Ehlert, 2016](#)). Nonetheless, Probst et al. (2017) obtained a positive association between T and reactive aggression when C levels were higher. As we explained above, in women, the menstrual cycle should be introduced as covariate because it tends to interfere with the association between variables. Moreover, in women, the secretion of T was substantially different from the amount liberated in men. Nonetheless, in studies that included both genders, they also found that higher C changes were related to high proneness to reactive aggression ([Böhnke et al., 2010a](#), [Gerra et al., 2001a](#), [Gerra et al., 2001b](#), [Gerra et al., 2004](#), [Gerra et al., 2007](#)). To fully understand this a priori contradictory result, it would be necessary to understand that those endocrine systems did not activate at the same time. Thus, the initial liberation of C to respond to a potential threat might be accompanied by an initial decrease of T (short-term), but a subsequent rise of their levels (mid-term). In fact, it should be kept in mind that most of these studies based their conclusions on salivary hormonal levels, which represent non-protein-bound serum hormone levels, but do not necessarily reflect those which directly interfere with brain networks ([Kaufman and Lamster, 2002](#)). Hence, this encourages us to revisit the model in order to accommodate several contexts, without viewing the association between these hormones and aggression as a static model, even including other hormones such as OX because their manipulation tends to promote decreases in reactive aggression ([Alcorn et al., 2015](#), [Berends et al., 2019](#), [Ne'eman et al., 2016](#), [Zhu et al., 2019b](#)). This last result could be explained by the effects of OX on facial emotion processing. In fact, it has been concluded that as OX levels

increase, the ability to decode emotions from facial expressions becomes better (Romero-Martínez et al., 2021). Therefore, a temporary rise of this hormone, might increase emotional decoding abilities, which in turn, might favor empathic abilities (e.g., increasing empathic concern about aggressive behavior consequences by interpreting facial signals) and consequently diminish reactive aggressive proneness.

We employed a broader perspective in order to include as many manuscripts as possible and to help determine whether reactive and proactive aggression are opposite or complementary forms of aggressive behavior. Based on the data included in this review, we can conclude that a large part of the research supports a complementary view. Specifically, genetic research indicated a relatively important overlap in their heritability, greatly modulated by non-shared environmental factors. Of the genetic factors, both present allelic variations that mainly affect serotonin transportation and monoaminergic degradation. The two forms also overlap in that the amygdala and temporal cortex were relatively important in understanding their proneness, even though they are mainly supported by relatively differentiated brain circuits. Finally, a low number of studies with psychophysiological variables concluded that a certain degree of hyperreactivity corresponds to reactive aggression, whereas hypoarousal represents proactive aggression.

This review has some limitations inherent to the review and to the research studies included. In comparison with a systematic review, a meta-analysis might restrain the number of studies by focusing on specific study designs to compute the potency of group differences. However, this might affect the richness of the conclusions, and so we decided to collect studies without considering their methodological robustness. In this regard, the majority of the studies included were cross-sectional with uncontrolled non-experimental designs (quasi-experiments), which might reduce the rigorousness of this review (see [Supplementary Table 1](#) for further details about the methodological quality of non-randomized studies). We are also concerned about the self-reports employed to assess aggression. As indicated in the introduction, most of them focus on the physical expression of both forms. Therefore, we also include computerized tasks to measure both forms of aggression. Unfortunately, there is an important bias in the data employing computerized tasks we collected because more studies assessed reactive aggression than proactive aggression. For instance, procedures such as TAP and PSAP were developed for measuring reactive aggression after provocation (Geniole et al., 2017, Taylor, 1967). In fact, the majority of the studies included in this review employed these tasks as a measurement of reactive aggression and only a minority included modified versions for assessing proactive aggression as well (attack without

provocation) (Chen, 2018; Riva et al., 2017; Sergiou et al., 2021; Weidler et al., 2020) or other types of tasks (Chester et al., 2019, Perach-Barzilay et al., 2013). The difference in number of publications only assessing reactive aggression instead of research combining procedures to measure both should be considered in future research to avoid potential biases in the interpretation of the data. It should even be highlighted that a large amount of research only paid attention to 5-HT, with less attention directed to other neurotransmitters, which were also important for aggressive behavior. Another limitation was the limited sample size in the studies included. Moreover, many of them did not control for potential type-1 error of multiple comparisons, the assessment of aggression was based on third parties (e.g., parents, teachers...), and there was a lack of control of multiple demographic factors acting as confounders, among others. In this sense, several studies pointed out the importance of including age for assessing aggressive behaviors. In fact, it has been demonstrated that scores tend to diminish with age (Fung et al., 2018, Vigil-Colet et al., 2015). A meta-analysis even signaled considerable variations in the effect of genetic common factors in reactive and proactive aggression heritability when they were assessed at different ages (Burt, 2009). Therefore, this should be considered a potential confounding factor and it would therefore be important to control it in future studies. Hence, these limitations increased the difficulty of obtaining unanimous conclusions. It should be noted that we only focused on the classical dichotomic model to categorize aggressive behavior in reactive vs proactive aggression (Dodge, 1991, Dodge and Coie, 1987), but we did not consider, for example, a subtype of proactive aggression such as appetitive (Elbert et al., 2010, Elbert et al., 2017, Elbert et al., 2018, Weierstall and Elbert, 2011). Due to the considerable psychobiological overlapping between reactive and proactive aggression, maybe the analysis of this subtype of proactive aggression, which tends to be extrinsically facilitated by the pleasure that the perpetration of aggression generates in contraposition to reactive aggression, might help to clearly establish a differentiated pattern of psychobiological underlying factors (e.g., allelic variants, brain networks...) for each type of aggression. Concretely, high activity polymorphisms Stin2 VNTR12 of the SLC6A4 gene seemed to be associated with high appetitive aggression, and the low activity polymorphisms with reactive aggression (Hemmings et al., 2018) Additionally, we also consider it particularly important to include in future research other concomitant factors of each type of aggression such as callous unemotional traits (e.g., lack or low empathic concern about the perpetration of aggression in others), which demonstrated being closely associated with proactive aggression (Werhahn et al., 2020) or impulsivity (Orri et al., 2020, Portnoy et al., 2014), among others, for clarifying the psychobiological distinction between these different modalities of aggression.

In summary, the present review demonstrated that reactive and proactive aggression share their etiological roots, but the environment modulates their expression as the individual develops. We also highlight the fact that our review not only offers background information about underlying biological bases of both types of aggression, but it also includes specific treatments to reduce their proneness (e.g., application of non-brain stimulation techniques over the PFC, pharmacological strategies with modulating effects on serotonergic neurotransmission, and oxytocin modulating effects). This increases the value of the conclusions for scholars and for clinical practitioners. In any case, we would like to reinforce the need for additional studies that measure proactive aggression and new instruments that make it possible to measure different dimensions (not only physical forms of aggression) of complex constructs related to reactive and proactive aggression.

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References

[Aggensteiner et al., 2020](#) P.M. Aggensteiner, N.E. Holz, B.W. Böttinger, S. Baumeister, S. Hohmann, J.E. Werhahn, D. Brandeis

The effects of callous-unemotional traits and aggression subtypes on amygdala activity in response to negative faces

Psychol. Med. (2020), pp. 1-9, [10.1017/S0033291720002111](https://doi.org/10.1017/S0033291720002111) ↗

[Google Scholar](#) ↗

[Ahmed et al., 2018](#) S.P. Ahmed, L.H. Somerville, C.L. Sebastian

Using temporal distancing to regulate emotion in adolescence: modulation by reactive aggression

Cogn. Emot., 32 (4) (2018), pp. 812-826, [10.1080/02699931.2017.1358698](https://doi.org/10.1080/02699931.2017.1358698) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Alcorn et al., 2015](#) J.L. Alcorn III, C.E. Green, J. Schmitz, S.D. Lane

Effects of oxytocin on aggressive responding in healthy adult males

Behav. Pharmacol., 26 (8 0 0) (2015), p. 798, [10.1097/FBP.0000000000000173](https://doi.org/10.1097/FBP.0000000000000173) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Anguelova et al., 2003](#) M. Anguelova, C. Benkelfat, G. Turecki

A systematic review of association studies investigating genes coding for serotonin receptors and the serotonin transporter: I. Affective disorders

Mol. Psychiatry, 8 (6) (2003), pp. 574-591, [10.1038/sj.mp.4001328](https://doi.org/10.1038/sj.mp.4001328) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Armstrong et al., 2021](#) T. Armstrong, J. Wells, D.L. Boisvert, R.H. Lewis, E.M. Cooke, M.

Woeckener, N. Kavish

An exploratory analysis of testosterone, cortisol, and aggressive behavior type in men and women

Biol. Psychol. (2021), Article 108073, [10.1016/j.biopsycho.2021.108073](https://doi.org/10.1016/j.biopsycho.2021.108073) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Armstrong et al., 2019](#) T. Armstrong, J. Wells, D.L. Boisvert, R. Lewis, E.M. Cooke, M.

Woeckener, N. Kavish

Skin conductance, heart rate and aggressive behavior type

Biol. Psychol., 141 (2019), pp. 44-51, [10.1016/j.biopsycho.2018.12.012](https://doi.org/10.1016/j.biopsycho.2018.12.012) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Assaad et al., 2006](#) J.M. Assaad, R.O. Pihl, J.R. Séguin, D. Nagin, F. Vitaro, R.E. Tremblay

Heart rate response to alcohol and intoxicated aggressive behavior

Alcohol.: Clin. Exp. Res., 30 (5) (2006), pp. 774-782, [10.1111/j.1530-0277.2006.00090.x](https://doi.org/10.1111/j.1530-0277.2006.00090.x) ↗

↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Baker et al., 2008](#) L.A. Baker, A. Raine, J. Liu, K.C. Jacobson

Differential genetic and environmental influences on reactive and proactive aggression in children

J. Abnorm. Child. Psychol., 36 (2008), pp. 1265-1278, [10.1007/s10802-008-9249-1](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Bakker-Huvenaars et al., 2020](#) M.J. Bakker-Huvenaars, C.U. Greven, P. Herpers, E. Wieggers, A. Jansen, R. Van Der Steen, J.K. Buitelaar

Saliva oxytocin, cortisol, and testosterone levels in adolescent boys with autism spectrum disorder, oppositional defiant disorder/conduct disorder and typically developing individuals

Eur. Neuropsychopharmacol., 30 (2020), pp. 87-101, [10.1016/j.euroneuro.2018.07.097](#) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Banny et al., 2014](#) A.M. Banny, W.L. Tseng, D. Murray-Close, C.E. Pitula, N.R. Crick

Borderline personality features as a predictor of forms and functions of aggression during middle childhood: Examining the roles of gender and physiological reactivity

Dev. Psychopathol., 26 (3) (2014), pp. 789-804, [10.1017/S095457941400039X](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Berends et al., 2019](#) Y.R. Berends, J.H. Tulen, A.I. Wierdsma, J. van Pelt, R. Feldman, O. Zagoory-Sharon, H.J. van Marle

Intranasal administration of oxytocin decreases task-related aggressive responses in healthy young males

Psychoneuroendocrinology, 106 (2019), pp. 147-154, [10.1016/j.psyneuen.2019.03.027](#) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Berman et al., 2009](#) M.E. Berman, M.S. McCloskey, J.R. Fanning, J.A. Schumacher, E.F. Coccaro
Serotonin augmentation reduces response to attack in aggressive individuals

Psychol. Sci., 20 (6) (2009), pp. 714-720, [10.1111/j.1467-9280.2009.02355.x](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Beyer et al., 2015](#) F. Beyer, T.F. Münte, M. Göttlich, U.M. Krämer

Orbitofrontal cortex reactivity to angry facial expression in a social interaction correlates with aggressive behavior

Cereb. Cortex, 25 (9) (2015), pp. 3057-3063, [10.1093/cercor/bhu101](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Beyer et al., 2014](#) F. Beyer, T.F. Münte, U.M. Krämer

Increased neural reactivity to socio-emotional stimuli links social exclusion and aggression

Biol. Psychol., 96 (2014), pp. 102-110, [10.1016/j.biopsycho.2013.12.008](#) ↗



[View PDF](#)

[View article](#)

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Bissonette and Roesch, 2016](#) G.B. Bissonette, M.R. Roesch

Development and function of the midbrain dopamine system: what we know and what we need to

Genes Brain Behav., 15 (1) (2016), pp. 62-73, [10.1111/gbb.12257](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Bobes et al., 2013](#) M.A. Bobes, F. Ostrosky, K. Diaz, C. Romero, K. Borja, Y. Santos, M. Valdés-Sosa

Linkage of functional and structural anomalies in the left amygdala of reactive-aggressive men

Soc. Cogn. Affect. Neurosci., 8 (8) (2013), pp. 928-936, [10.1093/scan/nss101](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Böhnke et al., 2010a](#) R. Böhnke, K. Bertsch, M.R. Kruk, S. Richter, E. Naumann

Exogenous cortisol enhances aggressive behavior in females, but not in males

Psychoneuroendocrinology, 35 (7) (2010), pp. 1034-1044,

[10.1016/j.psyneuen.2010.01.004](#) ↗



[View PDF](#)

[View article](#)

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Böhnke et al., 2010b](#) R. Böhnke, K. Bertsch, M.R. Kruk, E. Naumann

The relationship between basal and acute HPA axis activity and aggressive behavior in adults

J. Neural Transm., 117 (5) (2010), pp. 629-637, [10.1007/s00702-010-0391-x](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Brendgen et al., 2006](#) M. Brendgen, F. Vitaro, M. Boivin, G. Dionne, D. Pérois

Examining genetic and environmental effects on reactive versus proactive aggression

Dev. Psychol., 42 (6) (2006), p. 1299, [10.1037/0012-1649.42.6.1299](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Brown and Braver, 2005](#) J.W. Brown, T.S. Braver

Learned predictions of error likelihood in the anterior cingulate cortex
Science, 307 (5712) (2005), pp. 1118-1121, [10.1126/science.1105783](https://doi.org/10.1126/science.1105783) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Brzozowski et al., 2021](#) A. Brzozowski, S.M. Gillespie, L. Dixon, I.J. Mitchell
Cardiac autonomic function and psychological characteristics of
heterosexual female perpetrators of intimate partner physical aggression
J. Interpers. Violence, 36 (7–8) (2021), pp. 3638-3661, [10.1177/0886260518775748](https://doi.org/10.1177/0886260518775748) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Buades-Rotger et al., 2016](#) M. Buades-Rotger, C. Engelke, F. Beyer, B.G. Keevil, G. Brabant, U.M. Krämer
Endogenous testosterone is associated with lower amygdala reactivity to
angry faces and reduced aggressive behavior in healthy young women
Sci. Rep., 6 (1) (2016), pp. 1-14, [10.1038/srep38538](https://doi.org/10.1038/srep38538) ↗

[Google Scholar ↗](#)

[Buades-Rotger et al., 2019](#) M. Buades-Rotger, C. Engelke, U.M. Krämer
Trait and state patterns of basolateral amygdala connectivity at rest are
related to endogenous testosterone and aggression in healthy young
women
Brain Imaging Behav., 13 (2) (2019), pp. 564-576, [10.1007/s11682-018-9884-2](https://doi.org/10.1007/s11682-018-9884-2) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Buades-Rotger and Krämer, 2018](#) M. Buades-Rotger, U.M. Krämer
From words to action: Implicit attention to antisocial semantic cues
predicts aggression and amygdala reactivity to angry faces in healthy
young women
Aggress. Behav., 44 (6) (2018), pp. 624-637, [10.1002/ab.21787](https://doi.org/10.1002/ab.21787) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Campbell and Hausmann, 2013](#) A. Campbell, M. Hausmann
Effects of oxytocin on women's aggression depend on state anxiety
Aggress. Behav., 39 (4) (2013), pp. 316-322, [10.1002/ab.21478](https://doi.org/10.1002/ab.21478) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Carré et al., 2013](#) J.M. Carré, J.A. Campbell, E. Lozoya, S.M. Goetz, K.M. Welker
Changes in testosterone mediate the effect of winning on subsequent
aggressive behaviour

Psychoneuroendocrinology, 38 (10) (2013), pp. 2034-2041,
[10.1016/j.psyneuen.2013.03.008](https://doi.org/10.1016/j.psyneuen.2013.03.008) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Carré et al., 2017](#) J.M. Carré, S.N. Geniole, T.L. Ortiz, B.M. Bird, A. Videto, P.L. Bonin
Exogenous testosterone rapidly increases aggressive behavior in
dominant and impulsive men

Biol. Psychiatry, 82 (4) (2017), pp. 249-256, [10.1016/j.biopsych.2016.06.009](https://doi.org/10.1016/j.biopsych.2016.06.009) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Carré et al., 2010](#) J.M. Carré, J.D. Gilchrist, M.D. Morrissey, C.M. McCormick
Motivational and situational factors and the relationship between
testosterone dynamics and human aggression during competition
Biol. Psychol., 84 (2010), pp. 346-353, [10.1016/j.biopsycho.2010.04.001](https://doi.org/10.1016/j.biopsycho.2010.04.001) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Carré et al., 2014](#) J.M. Carré, A.M.R. Iselin, K.M. Welker, A.R. Hariri, K.A. Dodge
Testosterone reactivity to provocation mediates the effect of early
intervention on aggressive behavior

Psychol. Sci., 25 (5) (2014), pp. 1140-1146, [10.1177/0956797614525642](https://doi.org/10.1177/0956797614525642) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Carré and McCormick, 2008](#) J.M. Carré, C.M. McCormick
Aggressive behavior and change in salivary testosterone concentrations
predict willingness to engage in a competitive task

Horm. Behav., 54 (3) (2008), pp. 403-409, [10.1016/j.yhbeh.2008.04.008](https://doi.org/10.1016/j.yhbeh.2008.04.008) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Carré et al., 2009](#) J.M. Carré, S.K. Putnam, C.M. McCormick
Testosterone responses to competition predict future aggressive
behaviour at a cost to reward in men

Psychoneuroendocrinology, 34 (4) (2009), pp. 561-570, [10.1016/j.psyneuen.2008.10.018](https://doi.org/10.1016/j.psyneuen.2008.10.018)

↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Casat et al., 1995](#) C.D. Casat, D.A. Pearson, M.J. Van Davelaar, D.R. Cherek
Methylphenidate effects on a laboratory aggression measure in children
with ADHD

Psychopharmacol. Bull., 31 (2) (1995), pp. 353-356

[View in Scopus](#) ↗ [Google Scholar](#) ↗

- [Caspi et al., 2010](#) A. Caspi, A.R. Hariri, A. Holmes, R. Uher, T.E. Moffitt
Genetic sensitivity to the environment: the case of the serotonin transporter gene and its implications for studying complex diseases and traits
Am. J. Psychiatry, 167 (5) (2010), pp. 509-527, [10.1176/appi.ajp.2010.09101452](#) ↗
[Google Scholar](#) ↗
- [Caspi et al., 2003](#) A. Caspi, K. Sugden, T.E. Moffitt, A. Taylor, I.W. Craig, H. Harrington, R. Poulton
Influence of life stress on depression: moderation by a polymorphism in the 5-HTT gene
Science, 301 (5631) (2003), pp. 386-389, [10.1126/science.1083968](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Chen, 2018a](#) C.Y. Chen
Right ventrolateral prefrontal cortex involvement in proactive and reactive aggression: a transcranial direct current stimulation study
NeuroReport, 29 (17) (2018), pp. 1509-1515, [10.1097/WNR.0000000000001144](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Chen et al., 2019](#) C.Y. Chen, C.R. Chiou, C.H. Ko
Juveniles with a history of violent behavior show cognitive performance and electrophysiology consistent with inhibitory control and emotional feedback processing problems
Aggress. Behav., 45 (1) (2019), pp. 6-17, [10.1002/ab.21792](#) ↗
[Google Scholar](#) ↗
- [Chen et al., 2018b](#) F.R. Chen, A. Raine, D.A. Granger
Testosterone and proactive-reactive aggression in youth: the moderating role of harsh discipline
J. Abnorm. Child Psychol., 46 (8) (2018), pp. 1599-1612, [10.1007/s10802-018-0399-5](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Cherek and Lane, 2001](#) D.R. Cherek, S.D. Lane
Acute effects of D-fenfluramine on simultaneous measures of aggressive escape and impulsive responses of adult males with and without a history of conduct disorder
Psychopharmacology, 157 (3) (2001), pp. 221-227, [10.1007/s002130100812](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cherek and Lane, 1999](#) D.R. Cherek, S.D. Lane

Effects of d, l-fenfluramine on aggressive and impulsive responding in adult males with a history of conduct disorder

Psychopharmacology, 146 (4) (1999), pp. 473-481, [10.1007/pl00005493](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cherek et al., 2002b](#) D.R. Cherek, S.D. Lane, C.J. Pietras, J. Sharon, J.L. Steinberg

Acute effects of baclofen, a γ -aminobutyric acid-B agonist, on laboratory measures of aggressive and escape responses of adult male parolees with and without a history of conduct disorder

Psychopharmacology, 164 (2) (2002), pp. 160-167, [10.1007/s00213-002-1167-2](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cherek et al., 2002a](#) D.R. Cherek, S.D. Lane, C.J. Pietras, J.L. Steinberg

Effects of chronic paroxetine administration on measures of aggressive and impulsive responses of adult males with a history of conduct disorder

Psychopharmacology, 159 (3) (2002), pp. 266-274, [10.1007/s002130100915](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cherepkova et al., 2015](#) E.V. Cherepkova, V.N. Maksimov, L.I. Aftanas, P.N. Menshanov

Genotype and haplotype frequencies of the DRD4 VNTR polymorphism in the men with no history of ADHD, convicted of violent crimes

J. Crim. Justice, 43 (6) (2015), pp. 464-469, [10.1016/j.jcrimjus.2015.10.002](#) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Chester et al., 2019](#) D.S. Chester, S.B. Bell, C.N. DeWall, S.J. West, M. Romero-Lopez, A.W. Craig

Neural correlates of intertemporal choice in aggressive behavior

Aggress. Behav., 45 (5) (2019), pp. 507-516, [10.1002/ab.21838](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Coccaro et al., 1996](#) E.F. Coccaro, M.E. Berman, R.J. Kavoussi, R.L. Hauger

Relationship of prolactin response to d-fenfluramine to behavioral and questionnaire assessments of aggression in personality-disordered men

Biol. Psychiatry, 40 (3) (1996), pp. 157-164, [10.1016/0006-3223\(95\)00398-3](#) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Coccaro and Lee, 2019](#) E.F. Coccaro, R.J. Lee

5-HT_{2c} agonist, lorcaserin, reduces aggressive responding in
intermittent explosive disorder: a pilot study

Hum. Psychopharmacol.: Clin. Exp., 34 (6) (2019), Article e2714, [10.1002/hup.2714](https://doi.org/10.1002/hup.2714) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cote et al., 2013](#) K.A. Cote, C.M. McCormick, S.N. Geniole, R.P. Renn, S.D. MacAulay

Sleep deprivation lowers reactive aggression and testosterone in men

Biological Psychology, 92 (2) (2013), pp. 249-256, [10.1016/j.biopsycho.2012.09.011](https://doi.org/10.1016/j.biopsycho.2012.09.011) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cortes et al., 1988](#) R. Cortes, E. Soriano, A. Pazos, J.M. Probst

Palacios Autoradiography of antidepressant binding sites in the human
brain: localization using [3H] imipramine and [3H]

paroxetine Neuroscience, 27 (2) (1988), pp. 473-496



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Cohen, 1992](#) J. Cohen

Statistical power analysis

Curr. Dir. Psychol. Sci., 1 (3) (1992), pp. 98-101, [10.1111/1467-8721.ep10768783](https://doi.org/10.1111/1467-8721.ep10768783) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Craig et al., 2019](#) M.C. Craig, L.M. Mulder, M.P. Zwiers, A. Sethi, P.J. Hoekstra, A. Dietrich, J.
Naaijen

Distinct associations between fronto-striatal glutamate concentrations
and callous-unemotional traits and proactive aggression in disruptive
behavior

Cortex, 121 (2019), pp. 135-146, [10.1016/j.cortex.2019.08.017](https://doi.org/10.1016/j.cortex.2019.08.017) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Crespi, 2016](#) B.J. Crespi

Oxytocin, testosterone, and human social cognition

Biol. Rev., 91 (2) (2016), pp. 390-408, [10.1111/brv.12175](https://doi.org/10.1111/brv.12175) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Crozier et al., 2008](#) J.C. Crozier, K.A. Dodge, R.G. Fontaine, J.E. Lansford, J.E. Bates, G.S. Pettit,
R.W. Levenson

Social information processing and cardiac predictors of adolescent
antisocial behavior

J. Abnorm. Psychol., 117 (2) (2008), p. 253, [10.1037/0021-843X.117.2.253](https://doi.org/10.1037/0021-843X.117.2.253) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Dambacher et al., 2015a](#) F. Dambacher, A.T. Sack, J. Lobbestael, A. Arntz, S. Brugman, T. Schuhmann
Out of control: evidence for anterior insula involvement in motor impulsivity and reactive aggression
Soc. Cogn. Affect. Neurosci., 10 (4) (2015), pp. 508-516, [10.1093/scan/nsu077](#) ↗
[Google Scholar](#) ↗

[Dambacher et al., 2015b](#) F. Dambacher, T. Schuhmann, J. Lobbestael, A. Arntz, S. Brugman, A.T. Sack
Reducing proactive aggression through non-invasive brain stimulation
Soc. Cogn. Affect. Neurosci., 10 (10) (2015), pp. 1303-1309, [10.1093/scan/nsv018](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Dambacher et al., 2015c](#) F. Dambacher, T. Schuhmann, J. Lobbestael, A. Arntz, S. Brugman, A.T. Sack
No effects of bilateral tDCS over inferior frontal gyrus on response inhibition and aggression
PLoS One, 10 (7) (2015), Article e0132170, [10.1371/journal.pone.0132170](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Dekkers et al., 2019](#) T.J. Dekkers, J.A.A. van Rentergem, B. Meijer, A. Popma, E. Wagemaker, H.M. Huizenga
A meta-analytical evaluation of the dual-hormone hypothesis: does cortisol moderate the relationship between testosterone and status, dominance, risk taking, aggression, and psychopathy?
Neurosci. Biobehav. Rev., 96 (2019), pp. 250-271, [10.1016/j.neubiorev.2018.12.004](#) ↗
 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Denson et al., 2013](#) T.F. Denson, P.H. Mehta, D.H. Tan
Endogenous testosterone and cortisol jointly influence reactive aggression in women
Psychoneuroendocrinology, 38 (3) (2013), pp. 416-424, [10.1016/j.psyneuen.2012.07.003](#) ↗
 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Dodge, 1991](#) K.A. Dodge
The structure and function of reactive and proactive aggression
Dev. Treat. Child. Aggress., 16 (5) (1991), pp. 201-218, [10.1111/j.1467-6494.2009.00610.x](#) ↗

[Google Scholar ↗](#)

[Dodge and Coie, 1987](#) K.A. Dodge, J.D. Coie

Social-information-processing factors in reactive and proactive aggression in children's peer groups

J. Personal. Soc. Psychol., 53 (1987), pp. 1146-1158, [10.1037//0022-3514.53.6.1146 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Dougherty et al., 1999](#) D.M. Dougherty, J.M. Bjork, D.M. Marsh, F.G. Moeller

Influence of trait hostility on tryptophan depletion-induced laboratory aggression

Psychiatry Res., 88 (3) (1999), pp. 227-232

[https://doi.org/10.1016/s0165-1781\(99\)00088-8](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Dougherty et al., 1997](#) D.M. Dougherty, J.M. Bjork, F.G. Moeller, A.C. Swann

The influence of menstrual-cycle phase on the relationship between testosterone and aggression

Physiol. Behav., 62 (2) (1997), pp. 431-435

[https://doi.org/10.1016/s0031-9384\(97\)88991-3](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Eimontaite et al., 2019](#) I. Eimontaite, I. Schindler, M. De Marco, D. Duzzi, A. Venneri, V. Goel

Left amygdala and putamen activation modulate emotion driven decisions in the iterated Prisoner's Dilemma game

Front. Neurosci., 13 (2019), p. 741, [10.3389/fnins.2019.00741 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Elbert et al., 2017](#) T. Elbert, J.K. Moran, M. Schauer

Lust for violence: appetitive aggression as a fundamental part of human nature

e-Neuroforum, 23 (2) (2017), pp. 77-84, [10.1515/nf-2016-A056 ↗](#)

[Google Scholar ↗](#)

[Elbert et al., 2018](#) T. Elbert, M. Schauer, J.K. Moran

Two pedals drive the bi-cycle of violence: reactive and appetitive aggression

Curr. Opin. Psychol., 19 (2018), pp. 135-138, [10.1016/j.copsyc.2017.03.016 ↗](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Elbert et al., 2010](#) T. Elbert, R. Weierstall, M. Schauer

Fascination violence: On mind and brain of man hunters

Eur. Arch. Psychiatry Clin. Neurosci., 260 (2) (2010), pp. 100-105, [10.1007/s00406-010-0144-8](#) ↗

[Google Scholar](#) ↗

[Emmerling et al., 2016](#) F. Emmerling, T. Schuhmann, J. Lobbestael, A. Arntz, S. Brugman, A.T. Sack

The role of the insular cortex in retaliation

PLoS One, 11 (4) (2016), Article e0152000, [10.1371/journal.pone.0152000](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Engert and Pruessner, 2008](#) V. Engert, J.C. Pruessner

Dopaminergic and noradrenergic contributions to functionality in ADHD: the role of methylphenidate

Curr. Neuropharmacol., 6 (4) (2008), pp. 322-328, [10.2174/157015908787386069](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fanning et al., 2014](#) J.R. Fanning, M.E. Berman, C.R. Guillot, A. Marsic, M.S. McCloskey

Serotonin (5-HT) augmentation reduces provoked aggression associated with primary psychopathy traits

J. Personal. Disord., 28 (3) (2014), pp. 449-461, [10.1521/pedi_2012_26_065](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Farah et al., 2018](#) T. Farah, S. Ling, A. Raine, Y. Yang, R. Schug

Alexithymia and reactive aggression: the role of the amygdala

Psychiatry Res.: Neuroimaging, 281 (2018), pp. 85-91,

[10.1016/j.psychresns.2018.09.003](#) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fite et al., 2012](#) P.J. Fite, J.L. Rathert, L. Stoppelbein, L. Greening

Social problems as a mediator of the link between reactive aggression and withdrawn/depressed symptoms

J. Child Fam. Stud., 21 (2) (2012), pp. 184-189, [10.1007/s10826-011-9461-4](#) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fowler et al., 1987](#) J.S. Fowler, R.R. MacGregor, A.P. Wolf, C.D. Arnett, S.L. Dewey, D. Schlyer, B. Långström

Mapping human brain monoamine oxidase A and B with 11C-labeled suicide inactivators and PET

Science, 235 (4787) (1987), pp. 481-485, [10.1126/science.3099392](https://doi.org/10.1126/science.3099392) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fragkaki et al., 2019](#) I. Fragkaki, M. Cima, M. Verhagen, D.F. Maciejewski, M.P. Boks, P.A. Van Lier, W.H. Meeus

Oxytocin receptor gene (OXTR) and deviant peer affiliation: a gene–environment interaction in adolescent antisocial behavior

J. Youth Adolesc., 48 (1) (2019), pp. 86-101, [10.1007/s10964-018-0939-x](https://doi.org/10.1007/s10964-018-0939-x) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fullerton et al., 2019](#) A.F. Fullerton, N.J. Jackson, C. Tuvblad, A. Raine, L.A. Baker

Early childhood head injury attenuates declines in impulsivity and aggression across adolescent development in twins

Neuropsychology, 33 (8) (2019), p. 1035, [10.1037/neu0000570](https://doi.org/10.1037/neu0000570) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fulwiler et al., 2005](#) C. Fulwiler, J. Eckstine, S. Kalsy

Impulsive-aggressive traits, serotonin function, and alcohol-enhanced aggression

J. Clin. Pharmacol., 45 (1) (2005), pp. 94-100, [10.1177/0091270004270457](https://doi.org/10.1177/0091270004270457) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Fung et al., 2018](#) A.L.C. Fung, X. Li, M.J. Ramírez, B.Y.H. Lam, L. Millana, N.E. Fares-Otero

A cross-regional study of the reactive and proactive aggression of youth in Spain, Uruguay, mainland China, and Hong Kong

Soc. Dev., 27 (4) (2018), pp. 748-760, [10.1111/sode.12305](https://doi.org/10.1111/sode.12305) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gallardo-Pujol et al., 2013](#) D. Gallardo-Pujol, A. Andres-Pueyo, A. Maydeu-Olivares

MAOA genotype, social exclusion and aggression: an experimental test of a gene–environment interaction

Genes Brain Behav., 12 (2013), pp. 140-145, [10.1111/j.1601-183X.2012.00868](https://doi.org/10.1111/j.1601-183X.2012.00868) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gan et al., 2016](#) G. Gan, R.N. Preston-Campbell, S.J. Moeller, J.L. Steinberg, S.D. Lane, T. Maloney, N. Alia-Klein

Reward vs. retaliation—the role of the mesocorticolimbic salience network in human reactive aggression

Front. Behav. Neurosci., 10 (2016), p. 179, [10.1038/npp.2015.141](https://doi.org/10.1038/npp.2015.141) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

- [Gan et al., 2015](#) G. Gan, P. Sterzer, M. Marxen, U.S. Zimmermann, M.N. Smolka
Neural and behavioral correlates of alcohol-induced aggression under provocation
Neuropsychopharmacology, 40 (13) (2015), pp. 2886-2896, [10.3389/fnbeh.2016.00179](#)
[↗](#)
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [Gao et al., 2018](#) Y. Gao, K. Mendez, X. Li, M.C. Wang
Autonomic conditioning to monetary and social stimuli and aggression in children
Aggress. Behav., 44 (2) (2018), pp. 147-155, [10.1002/ab.21738](#) [↗](#)
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [Gao et al., 2015](#) Y. Gao, C. Tuvblad, A. Schell, L. Baker, A. Raine
Skin conductance fear conditioning impairments and aggression: a longitudinal study
Psychophysiology, 52 (2) (2015), pp. 288-295, [10.1111/psyp.12322](#) [↗](#)
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [Geniole et al., 2013](#) S.N. Geniole, M.A. Busseri, C.M. McCormick
Testosterone dynamics and psychopathic personality traits independently predict antagonistic behavior towards the perceived loser of a competitive interaction
Horm. Behav., 64 (5) (2013), pp. 790-798, [10.1016/j.yhbeh.2013.09.005](#) [↗](#)
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [Geniole et al., 2011](#) S.N. Geniole, J.M. Carré, C.M. McCormick
State, not trait, neuroendocrine function predicts costly reactive aggression in men after social exclusion and inclusion
Biol. Psychol., 87 (1) (2011), pp. 137-145, [10.1016/j.biopsycho.2011.02.020](#) [↗](#)
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [Geniole et al., 2017](#) S.N. Geniole, E.T. MacDonell, C.M. McCormick
The point subtraction aggression paradigm as a laboratory tool for investigating the neuroendocrinology of aggression and competition
Horm. Behav., 92 (2017), pp. 103-116, [10.1016/j.yhbeh.2016.04.006](#) [↗](#)
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [Gerra et al., 2007](#) G. Gerra, A. Zaimovic, M.A. Raggi, G. Moi, B. Branchi, M. Moroni, F. Brambilla

Experimentally induced aggressiveness in heroin-dependent patients treated with buprenorphine: comparison of patients receiving methadone and healthy subjects

Psychiatry Res., 149 (1–3) (2007), pp. 201-213, [10.1016/j.psychres.2006.02.013](https://doi.org/10.1016/j.psychres.2006.02.013) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gerra et al., 2004](#) G. Gerra, A. Zaimovic, G. Moi, M. Bussandri, C. Bubici, M. Mossini, F. Brambilla

Aggressive responding in abstinent heroin addicts: neuroendocrine and personality correlates

Prog. Neuro-Psychopharmacol. Biol. Psychiatry, 28 (1) (2004), pp. 129-139,

[10.1016/j.pnpbp.2003.09.029](https://doi.org/10.1016/j.pnpbp.2003.09.029) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gerra et al., 2001b](#) G. Gerra, A. Zaimovic, R. Ampollini, F. Giusti, R. Delsignore, M.A. Raggi, F. Brambilla

Experimentally induced aggressive behavior in subjects with 3, 4-methylenedioxy-methamphetamine (“Ecstasy”) use history: psychobiological correlates

J. Subst. Abus., 13 (4) (2001), pp. 471-491, [10.1016/s0899-3289\(01\)00094-3](https://doi.org/10.1016/s0899-3289(01)00094-3) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gerra et al., 2001a](#) G. Gerra, A. Zaimovic, M.A. Raggi, F. Giusti, R. Delsignore, S. Bertacca, F. Brambilla

Aggressive responding of male heroin addicts under methadone treatment: psychometric and neuroendocrine correlates

Drug Alcohol Depend., 65 (1) (2001), pp. 85-95, [10.1016/s0376-8716\(01\)00152-1](https://doi.org/10.1016/s0376-8716(01)00152-1) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gilam et al., 2018](#) G. Gilam, R. Abend, G. Gurevitch, A. Erdman, H. Baker, Z. Ben-Zion, T. Hendler
Attenuating anger and aggression with neuromodulation of the vmPFC: a simultaneous tDCS-fMRI study

Cortex, 109 (2018), pp. 156-170, [10.1016/j.cortex.2018.09.010](https://doi.org/10.1016/j.cortex.2018.09.010) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Glenn et al., 2019](#) A.L. Glenn, J.E. Lochman, T. Dishion, N.P. Powell, C. Boxmeyer, F. Kassing, D. Romero

Toward tailored interventions: sympathetic and parasympathetic functioning predicts responses to an intervention for conduct problems

delivered in two formats

Prev. Sci., 20 (1) (2019), pp. 30-40, [10.1007/s11121-017-0859-0](https://doi.org/10.1007/s11121-017-0859-0) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gowin et al., 2013](#) J.L. Gowin, C.E. Green, J.L. Alcorn, A.C. Swann, F.G. Moeller, S.D. Lane

The role of cortisol and psychopathy in the cycle of violence

Psychopharmacology, 227 (4) (2013), pp. 661-672, [10.1007/s00213-013-2992-1](https://doi.org/10.1007/s00213-013-2992-1) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Gowin et al., 2010](#) J.L. Gowin, A.C. Swann, F.G. Moeller, S.D. Lane

Zolmitriptan and human aggression: interaction with alcohol

Psychopharmacology, 210 (4) (2010), pp. 521-531, [10.1007/s00213-010-1851-6](https://doi.org/10.1007/s00213-010-1851-6) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Hagan et al., 2020](#) M.J. Hagan, S.F. Waters, S. Holley, L. Moctezuma, M. Gentry

The interactive effect of family conflict history and physiological reactivity on different forms of aggression in young women

Biol. Psychol., 153 (2020), Article 107888, [10.1016/j.biopsycho.2020.107888](https://doi.org/10.1016/j.biopsycho.2020.107888) ↗

 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Hemmings et al., 2018](#) S.M.J. Hemmings, K. Xulu, J. Sommer, M. Hinsberger, S. Malan-Muller, G. Tromp, S. Seedat

Appetitive and reactive aggression are differentially associated with the STin2 genetic variant in the serotonin transporter gene

Sci. Rep., 8 (1) (2018), pp. 1-9, [10.1038/s41598-018-25066-8](https://doi.org/10.1038/s41598-018-25066-8) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Higgins et al., 2019](#) J.P. Higgins, J. Thomas, J. Chandler, M. Cumpston, T. Li, M.J. Page, V.A. Welch

Cochrane Handbook for Systematic Reviews of Interventions

John Wiley & Sons, Chichester (UK) (2019)

[Google Scholar](#) ↗

[Hoaken et al., 2003](#) P.N. Hoaken, T. Campbell, S.H. Stewart, R.O. Pihl

Effects of alcohol on cardiovascular reactivity and the mediation of aggressive behaviour in adult men and women

Alcohol. Alcohol., 38 (1) (2003), pp. 84-92, [10.1093/alcalc/agg022](https://doi.org/10.1093/alcalc/agg022) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Hoffer et al., 2018](#) T. Hoffer, H. Hargreaves-Cormany, Y. Muirhead, J.R. Meloy

Meloy's bimodal theory of affective (reactive) and predatory (instrumental) violence

Violence in Animal Cruelty Offenders, Springer, Cham (2018), pp. 21-23

[Crossref ↗](#) [Google Scholar ↗](#)

[Hofhansel et al., 2020](#) L. Hofhansel, C. Weidler, M. Votinov, B. Clemens, A. Raine, U. Habel
Morphology of the criminal brain: gray matter reductions are linked to antisocial behavior in offenders

Brain Struct. Funct., 225 (7) (2020), pp. 2017-2028, [10.1007/s00429-020-02106-6 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Horvitz, 2000](#) J.C. Horvitz

Mesolimbocortical and nigrostriatal dopamine responses to salient non-reward events

Neuroscience, 96 (4) (2000), pp. 651-656, [10.1016/S0306-4522\(00\)00019-1 ↗](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Hubbard et al., 2002](#) J.A. Hubbard, C.M. Smithmyer, S.R. Ramsden, E.H. Parker, K.D. Flanagan, K.F. Dearing, R.F. Simons

Observational, physiological, and self-report measures of children's anger: Relations to reactive versus proactive aggression

Child Dev., 73 (4) (2002), pp. 1101-1118, [10.1111/1467-8624.00460 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Jiang et al., 2018b](#) Q. Jiang, H.O.U. Lulu, Q.I.U. Jiang, L.I. Changran, W.A.N.G. Huanzhen

The relationship between the caudate nucleus-orbitomedial prefrontal cortex connectivity and reactive aggression: a resting-state fMRI study

Acta Psychol. Sin., 50 (6) (2018), pp. 655-666, [10.3724/SP.J.1041.2018.00655 ↗](#)

[Google Scholar ↗](#)

[Juujärvi et al., 2006](#) P. Juujärvi, J. Kaartinen, T. Laitinen, E. Vanninen, L. Pulkkinen

Effects of physical provocations on heart rate reactivity and reactive aggression in children

Aggress. Behav., 32 (2) (2006), pp. 99-109, [10.1002/ab.20129 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kaltsouni et al., 2021](#) E. Kaltsouni, P.M. Fisher, M. Dubol, S. Hustad, R. Lanzenberger, V.G.

Frokjaer, I. Sundström-Poromaa

Brain reactivity during aggressive response in women with premenstrual dysphoric disorder treated with a selective progesterone

receptor modulator

Neuropsychopharmacology, 46 (8) (2021), pp. 1460-1467, [10.1038/s41386-021-01010-9](https://doi.org/10.1038/s41386-021-01010-9)

[↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kassing et al., 2018](#) F. Kassing, J.E. Lochman, A.L. Glenn

Autonomic functioning in reactive versus proactive aggression: the influential role of inconsistent parenting

Aggress. Behav., 44 (5) (2018), pp. 524-536, [10.1002/ab.21772](https://doi.org/10.1002/ab.21772) [↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kaufman and Lamster, 2002](#) E. Kaufman, I.B. Lamster

The diagnostic applications of saliva—a review

Crit. Rev. Oral Biol. Med., 13 (2) (2002), pp. 197-212, [10.1177/154411130201300209](https://doi.org/10.1177/154411130201300209) [↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kötting et al., 2013](#) W.F. Kötting, S. Bubenzer, K. Helmbold, A. Eisert, T.J. Gaber, F.D. Zepf

Effects of tryptophan depletion on reactive aggression and aggressive decision-making in young people with ADHD

Acta Psychiatr. Scand., 128 (2) (2013), pp. 114-123, [10.1111/acps.12001](https://doi.org/10.1111/acps.12001) [↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kolla et al., 2014](#) N.J. Kolla, S. Attard, G. Craig, N. Blackwood, S. Hodgins

Monoamine oxidase A alleles in violent offenders with antisocial personality disorder: high activity associated with proactive aggression

Crim. Behav. Ment. Health, 24 (2014), p. 368, [10.1002/cbm.1917](https://doi.org/10.1002/cbm.1917) [↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kolla et al., 2021](#) N.J. Kolla, I. Boileau, K. Karas, J.J. Watts, P. Rusjan, S. Houle, R. Mizrahi

Lower amygdala fatty acid amide hydrolase in violent offenders with antisocial personality disorder: an [11C] CURB positron emission tomography study

Transl. Psychiatry, 11 (1) (2021), pp. 1-11, [10.1038/s41398-020-01144-2](https://doi.org/10.1038/s41398-020-01144-2) [↗](#)

[Google Scholar ↗](#)

[Kolla et al., 2018](#) N.J. Kolla, K. Dunlop, J.H. Meyer, J. Downar

Corticostriatal connectivity in antisocial personality disorder by MAO-A genotype and its relationship to aggressive behavior

Int. J. Neuropsychopharmacol., 21 (8) (2018), pp. 725-733, [10.1093/ijnp/pyy035](https://doi.org/10.1093/ijnp/pyy035) [↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Krämer et al., 2011](#) U.M. Krämer, J. Riba, S. Richter, T.F. Münte

An fMRI study on the role of serotonin in reactive aggression

PLoS One, 6 (11) (2011), Article e27668, [10.1371/journal.pone.0027668](https://doi.org/10.1371/journal.pone.0027668) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Kuepper et al., 2013](#) Y. Kuepper, P. Grant, C. Wielpuetz, J. Hennig

MAOA-uVNTR genotype predicts interindividual differences in experimental aggressiveness as a function of the degree of provocation

Behav. Brain Res., 247 (2013), pp. 73-78, [10.1016/j.bbr.2013.03.002](https://doi.org/10.1016/j.bbr.2013.03.002) ↗

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Lazebny et al., 2020](#) O.E. Lazebny, A.M. Kulikov, P.R. Butovskaya, P.A. Proshakov, A.V. Fokin, M.L. Butovskaya

Analysis of aggressive behavior in young Russian males using 250 SNP markers

Russ. J. Genet., 56 (9) (2020), pp. 1118-1128, [10.1134/S1022795420080098](https://doi.org/10.1134/S1022795420080098) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Lee and Ham, 2008](#) B.T. Lee, B.J. Ham

Monoamine oxidase A-uVNTR genotype affects limbic brain activity in response to affective facial stimuli

Neuroreport, 19 (5) (2008), pp. 515-519, [10.1097/WNR.0b013e3282f94294](https://doi.org/10.1097/WNR.0b013e3282f94294) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Leech and Sharp, 2014](#) R. Leech, D.J. Sharp

The role of the posterior cingulate cortex in cognition and disease

Brain, 137 (1) (2014), pp. 12-32, [10.1093/brain/awt162](https://doi.org/10.1093/brain/awt162) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Ling et al., 2020](#) S. Ling, A. Raine, O. Choy, R. Hamilton

Effects of prefrontal cortical stimulation on aggressive and antisocial behavior: a double-blind, stratified, randomized, sham-controlled, parallel-group trial

J. Exp. Criminol., 16 (2020), pp. 367-387, [10.1007/s11292-020-09427-w](https://doi.org/10.1007/s11292-020-09427-w) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Liu et al., 2017](#) J. Liu, R. Zhong, W. Xiong, H. Liu, C. Eisenegger, X. Zhou

Melatonin increases reactive aggression in humans

Psychopharmacology, 234 (19) (2017), pp. 2971-2978, [10.1007/s00213-017-4693-7](https://doi.org/10.1007/s00213-017-4693-7) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Little et al., 2003](#) T.D. Little, S.M. Jones, C.C. Henrich, P.H. Hawley

Disentangling the “whys” from the “whats” of aggressive behavior

International Journal of Behavioral Development, 27 (2003), pp. 122-183

[Crossref ↗](#) [Google Scholar ↗](#)

[Lopez-Duran et al., 2009](#) N.L. Lopez-Duran, S.L. Olson, N.J. Hajal, B.T. Felt, D.M. Vazquez

Hypothalamic pituitary adrenal axis functioning in reactive and proactive aggression in children

J. Abnorm. Child Psychol., 37 (2009), pp. 169-182, [10.1007/s10802-008-9263-3 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Lotze et al., 2007](#) M. Lotze, R. Veit, S. Anders, N. Birbaumer

Evidence for a different role of the ventral and dorsal medial prefrontal cortex for social reactive aggression: An interactive fMRI study

Neuro Image, 34 (1) (2007), pp. 470-478, [10.1016/j.neuroimage.2006.09.028 ↗](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Lozier et al., 2014](#) L.M. Lozier, E.M. Cardinale, J.W. VanMeter, A.A. Marsh

Mediation of the relationship between callous-unemotional traits and proactive aggression by amygdala response to fear among children with conduct problems

JAMA Psychiatry, 71 (6) (2014), pp. 627-636, [10.1001/jamapsychiatry.2013.4540 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Luppino et al., 2014](#) D. Luppino, C. Moul, D.J. Hawes, J. Brennan, M.R. Dadds

Association between a polymorphism of the vasopressin 1B receptor gene and aggression in children

Psychiatr. Genet., 24 (5) (2014), pp. 185-190, [10.1097/YPG.0000000000000036 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[MacDonald et al., 2019](#) K.J. MacDonald, K.A. Lustig, S.N. Geniole, C.M. McCormick, K.A. Cote

Sleep restriction alters reactive aggressive behavior and its relationship with sex hormones

Aggress. Behav., 45 (2) (2019), pp. 193-205, [10.1002/ab.21809 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Mäki-Marttunen et al., 2019](#) V. Mäki-Marttunen, T. Hagen, T. Espeseth

Proactive and reactive modes of cognitive control can operate independently and simultaneously

Acta Psychol., 199 (2019), Article 102891, [10.1016/j.actpsy.2019.102891](https://doi.org/10.1016/j.actpsy.2019.102891) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Martin et al., 1978](#) N.G. Martin, L.J. Eaves, M.J. Kearsley, P. Davies

The power of the classical twin study

Heredity, 40 (1) (1978), pp. 97-116, [10.1038/hdy.1978.10](https://doi.org/10.1038/hdy.1978.10) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Martín-Ramírez, 2010](#) J. Martín-Ramírez

The usefulness of distinguishing types of aggression by function

Int. Soc. Sci. J., 61 (200–201) (2010), pp. 263-272, [10.1111/j.1468-2451.2011.01762.x](https://doi.org/10.1111/j.1468-2451.2011.01762.x) ↗

[Google Scholar](#) ↗

[Martín-Ramírez and Andreu, 2006](#) J. Martín-Ramírez, J.M. Andreu

Aggression, and some related psychological constructs (anger, hostility, and impulsivity) Some comments from a research project

Neurosci. Biobehav. Rev., 30 (3) (2006), pp. 276-291, [10.1016/j.neubiorev.2005.04.015](https://doi.org/10.1016/j.neubiorev.2005.04.015) ↗

[Google Scholar](#) ↗

[McCloskey et al., 2009](#) M.S. McCloskey, M.E. Berman, D.J. Echevarria, E.F. Coccaro

Effects of acute alcohol intoxication and paroxetine on aggression in men

Alcohol.: Clin. Exp. Res., 33 (4) (2009), pp. 581-590, [10.1111/j.1530-0277.2008.00872.x](https://doi.org/10.1111/j.1530-0277.2008.00872.x) ↗

↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Meloy, 2006](#) J. Meloy

Empirical basis and forensic application of affective and predatory violence

Aust. N. Z. J. Psychiatry, 40 (6) (2006), pp. 539-547, [10.1080/j.1440-1614.2006.01837.x](https://doi.org/10.1080/j.1440-1614.2006.01837.x) ↗

↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Meyer-Lindenberg et al., 2006](#) A. Meyer-Lindenberg, J.W. Buckholtz, B. Kolachana, A.R. Hariri, L. Pezawas, G. Blasi, D.R. Weinberger

Neural mechanisms of genetic risk for impulsivity and violence in humans

Proc. Natl. Acad. Sci. U.S.A., 103 (16) (2006), pp. 6269-6274, [10.1073/pnas.0511311103](https://doi.org/10.1073/pnas.0511311103) ↗

↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Michalska et al., 2016](#) K.J. Michalska, T.A. Zeffiro, J. Decety

Brain response to viewing others being harmed in children with conduct disorder symptoms

J. Child Psychol. Psychiatry, 57 (4) (2016), pp. 510-519, [10.1111/jcpp.12474 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Moeller et al., 1996](#) F.G. Moeller, D.M. Dougherty, A.C. Swann, D. Collins, C.M. Davis, D.R. Cherek

Tryptophan depletion and aggressive responding in healthy males

Psychopharmacology, 126 (2) (1996), pp. 97-103, [10.1007/BF02246343 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Moher et al., 2009](#) D. Moher, A. Liberati, J. Tetzlaff, D.G. Altman, Prisma Group

Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement

PLoS Med., 6 (7) (2009), Article e1000097, [10.1371/journal.pmed.1000097 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Moore et al., 2018](#) C.C. Moore, J. Hubbard, M.T. Morrow, L.R. Barhight, M.M. Lines, M. Sallee, C.T. Hyde

The simultaneous assessment of and relations between children's sympathetic and parasympathetic psychophysiology and their reactive and proactive aggression

Aggress. Behav., 44 (6) (2018), pp. 614-623, [10.1002/ab.21786 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Muñoz et al., 2008](#) L.C. Muñoz, P.J. Frick, E.R. Kimonis, K.J. Aucoin

Types of aggression, responsiveness to provocation, and callous-unemotional traits in detained adolescents

J. Abnorm. Child Psychol., 36 (1) (2008), pp. 15-28, [10.1007/s10802-007-9137-0 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Murray-Close et al., 2017](#) D. Murray-Close, L.A. Holterman, N.L. Breslend, A. Sullivan

Psychophysiology of proactive and reactive relational aggression

Biol. Psychol., 130 (2017), pp. 77-85, [10.1016/j.biopsycho.2017.10.005 ↗](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Murray-Close and Rellini, 2012](#) D. Murray-Close, A.H. Rellini

Cardiovascular reactivity and proactive and reactive relational aggression among women with and without a history of sexual abuse
Biol. Psychol., 89 (1) (2012), pp. 54-62, [10.1016/j.biopsycho.2011.09.008](https://doi.org/10.1016/j.biopsycho.2011.09.008) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Naaijen et al., 2020](#) J. Naaijen, L.M. Mulder, S. Ilbegi, S. de Bruijn, R. Kleine-Deters, A. Dietrich, J.K. Buitelaar

Specific cortical and subcortical alterations for reactive and proactive aggression in children and adolescents with disruptive behavior
Neuroimage: Clin., 27 (2020), Article 102344, [10.1016/j.nicl.2020.102344](https://doi.org/10.1016/j.nicl.2020.102344) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Ne'eman et al., 2016](#) R. Ne'eman, N. Perach-Barzilay, M. Fischer-Shofty, A. Atias, S.G. Shamay-Tsoory

Intranasal administration of oxytocin increases human aggressive behavior

Horm. Behav., 80 (2016), pp. 125-131, [10.1016/j.yhbeh.2016.01.015](https://doi.org/10.1016/j.yhbeh.2016.01.015) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Orri et al., 2020](#) M. Orri, M.C. Geoffroy, G. Turecki, B. Feng, M. Brendgen, F. Vitaro, M. Boivin
Contribution of genes and environment to the longitudinal association between childhood impulsive-aggression and suicidality in adolescence
J. Child Psychol. Psychiatry, 61 (6) (2020), pp. 711-720, [10.1111/jcpp.13163](https://doi.org/10.1111/jcpp.13163) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Paquin et al., 2014](#) S. Paquin, E. Lacourse, M. Brendgen, F. Vitaro, G. Dionne, R.E. Tremblay, M. Boivin

The genetic-environmental architecture of proactive and reactive aggression throughout childhood

Mon. für Kriminol. und Strafr., 97 (5–6) (2014), pp. 398-420, [10.1515/mks-2014-975-609](https://doi.org/10.1515/mks-2014-975-609) ↗

[Google Scholar](#) ↗

[Paquin et al., 2017](#) S. Paquin, E. Lacourse, M. Brendgen, F. Vitaro, G. Dionne, R.E. Tremblay, M. Boivin

Heterogeneity in the development of proactive and reactive aggression in childhood: Common and specific genetic-environmental factors

PLoS One, 12 (12) (2017), Article e0188730, [10.1371/journal.pone.0188730](https://doi.org/10.1371/journal.pone.0188730) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Pardini et al., 2014](#) D.A. Pardini, A. Raine, K. Erickson, R. Loeber

Lower amygdala volume in men is associated with childhood aggression, early psychopathic traits, and future violence

Biol. Psychiatry, 75 (1) (2014), pp. 73-80, [10.1016/j.biopsych.2013.04.003](#) ↗



[View PDF](#)

[View article](#)

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Peeters et al., 2020](#) D.G. Peeters, W.G. Lange, A.K.L. Von Borries, B. Franke, I. Volman, J.R.

Homberg, K. Roelofs

Threat-avoidance tendencies moderate the link between serotonin transporter genetic variation and reactive aggression

Front. Behav. Neurosci., 14 (2020), [10.3389/fnbeh.2020.562098](#) ↗

[Google Scholar](#) ↗

[Perach-Barzilay et al., 2013](#) N. Perach-Barzilay, A. Tauber, E. Klein, A. Chistyakov, R. Ne'Eman,

S.G. Shamay-Tsoory

Asymmetry in the dorsolateral prefrontal cortex and aggressive behavior: a continuous theta-burst magnetic stimulation study

Soc. Neurosci., 8 (2) (2013), pp. 178-188, [10.1080/17470919.2012.720602](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Peterson and Brown, 2005](#) R.A. Peterson, S.P. Brown

On the use of beta coefficients in meta-analysis

J. Appl. Psychol., 90 (1) (2005), p. 175, [10.1037/0021-9010.90.1.175](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Pincham et al., 2015](#) H.L. Pincham, C. Wu, C. Killikelly, L. Vuillier, R.P. Fearon

Social provocation modulates decision making and feedback processing: examining the trajectory of development in adolescent participants

Dev. Cogn. Neurosci., 15 (2015), pp. 58-66, [10.1016/j.dcn.2015.10.003](#) ↗



[View PDF](#)

[View article](#)

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Polman et al., 2007](#) H. Polman, B.O. de Castro, W. Koops, H.W. van Boxtel, W.W. Merk

A metaanalysis of the distinction between reactive and proactive aggression in children and adolescents

J. Abnorm. Child Psychol., 35 (2007), pp. 522-535, [10.1007/s10802-007-9109-4](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Porges, 2011](#) Porges, S.W. (2011). The polyvagal theory. New York: Norton.

[Google Scholar](#) ↗

- [Pfundmair et al., 2018](#) M. Pfundmair, A. Reinelt, C.N. DeWall, L. Feldmann
Oxytocin strengthens the link between provocation and aggression among low anxiety people
Psychoneuroendocrinology, 93 (2018), pp. 124-132, [10.1016/j.psyneuen.2018.04.025](#) ↗
 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Poltavski et al., 2018](#) D. Poltavski, R. Van Eck, A.T. Winger, C. Honts
Using a polygraph system for evaluation of the social desirability response bias in self-report measures of aggression
Appl. Psychophysiol. Biofeedback, 43 (4) (2018), pp. 309-318, [10.1007/s10484-018-9414-4](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Pope et al., 2000](#) H.G. Pope, E.M. Kouri, J.I. Hudson
Effects of supraphysiologic doses of testosterone on mood and aggression in normal men: a randomized controlled trial
Arch. Gen. Psychiatry, 57 (2) (2000), pp. 133-140, [10.1001/archpsyc.57.2.133](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Portnoy et al., 2014](#) J. Portnoy, A. Raine, F.R. Chen, D. Pardini, R. Loeber, J.R. Jennings
Heart rate and antisocial behavior: The mediating role of impulsive sensation seeking
Criminology, 52 (2) (2014), pp. 292-311, [10.1111/1745-9125.12038](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Poustka et al., 2010](#) L. Poustka, A. Maras, E. Hohm, J. Feller, M. Holtmann, T. Banaschewski, M. Laucht
Negative association between plasma cortisol levels and aggression in a high-risk community sample of adolescents
J. Neural Transm., 117 (5) (2010), pp. 621-627, [10.1007/s00702-010-0386-7](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Probst et al., 2018](#) F. Probst, J. Golle, V. Lory, J.S. Lobmaier
Reactive aggression tracks within-participant changes in women's salivary testosterone
Aggress. Behav., 44 (4) (2018), pp. 362-371, [10.1002/ab.21757](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Puhalla et al., 2020a](#) A.A. Puhalla, D.A. Kulper, M.K. Fahlgren, M.S. McCloskey

The relationship between resting heart rate variability, hostility, and in vivo aggression among young adults

J. Aggress. Maltreatment Trauma, 29 (2) (2020), pp. 206-222,

[10.1080/10926771.2018.1558324](https://doi.org/10.1080/10926771.2018.1558324) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Puhalla and McCloskey, 2020b](#) A.A. Puhalla, M.S. McCloskey

The relationship between physiological reactivity to provocation and emotion dysregulation with proactive and reactive aggression

Biol. Psychol., 155 (2020), Article 107931, [10.1016/j.biopsycho.2020.107931](https://doi.org/10.1016/j.biopsycho.2020.107931) ↗



[View PDF](#)

[View article](#)

[View in Scopus ↗](#)

[Google Scholar ↗](#)

[Puiu et al., 2018](#) A.A. Puiu, O. Wudarczyk, K.S. Goerlich, M. Votinov, B. Herpertz-Dahlmann, B. Turetsky, K. Konrad

Impulsive aggression and response inhibition in attention-deficit/hyperactivity disorder and disruptive behavioral disorders: Findings from a systematic review

Neurosci. Biobehav. Rev., 90 (2018), pp. 231-246, [10.1016/j.neubiorev.2018.04.016](https://doi.org/10.1016/j.neubiorev.2018.04.016) ↗



[View PDF](#)

[View article](#)

[View in Scopus ↗](#)

[Google Scholar ↗](#)

[Raine et al., 2014](#) A. Raine, A.L.C. Fung, J. Portnoy, O. Choy, V.L. Spring

Low heart rate as a risk factor for child and adolescent proactive aggressive and impulsive psychopathic behavior

Aggress. Behav., 40 (4) (2014), pp. 290-299, [10.1002/ab.21523](https://doi.org/10.1002/ab.21523) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Raine et al., 2006](#) A. Raine, K. Dodge, R. Loeber, L. Gatzke-Kopp, D. Lynam, C. Reynolds, J. Liu
The reactive–proactive aggression questionnaire: Differential correlates of reactive and proactive aggression in adolescent boys

Aggressive Behavior: Official Journal of the International Society for Research on Aggression, 32 (2) (2006), pp. 159-171

[Crossref ↗](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Repple et al., 2018](#) J. Repple, U. Habel, L. Wagels, C.M. Pawliczek, F. Schneider, N. Kohn
Sex differences in the neural correlates of aggression

Brain Struct. Funct., 223 (9) (2018), pp. 4115-4124, [10.1007/s00429-018-1739-5](https://doi.org/10.1007/s00429-018-1739-5) ↗

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Richardson, 2011](#) J.T. Richardson

Eta squared and partial eta squared as measures of effect size in educational research

Educ. Res. Rev., 6 (2) (2011), pp. 135-147, [10.1016/j.edurev.2010.12.001](https://doi.org/10.1016/j.edurev.2010.12.001) ↗



[View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗

[Rinnewitz et al., 2019](#) L. Rinnewitz, P. Parzer, J. Koenig, K. Bertsch, R. Brunner, F. Resch, M. Kaess

A biobehavioral validation of the Taylor aggression paradigm in female adolescents

Sci. Rep., 9 (1) (2019), pp. 1-10, [10.1038/s41598-019-43456-4](https://doi.org/10.1038/s41598-019-43456-4) ↗

[Google Scholar](#) ↗

[Romero-Martínez et al., 2019](#) Á. Romero-Martínez, S. Murciano-Martí, L. Moya-Albiol

Is sertraline a good pharmacological strategy to control anger? results of a systematic review

Behav. Sci., 9 (5) (2019), p. 57, [10.3390/bs9050057](https://doi.org/10.3390/bs9050057) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Romero-Martínez et al., 2021](#) Á. Romero-Martínez, C. Sarrate-Costa, L. Moya-Albiol

A systematic review of the role of oxytocin, cortisol, and testosterone in facial emotional processing

Biology, 10 (12) (2021), p. 1334, [10.3390/biology10121334](https://doi.org/10.3390/biology10121334) ↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Moya-Albiol, L. & Romero-Martínez, Á. \(2020\). Neurocriminología. Madrid: Piramide.](#) Moya-Albiol, L. & Romero-Martínez, Á. (2020). Neurocriminología. Madrid: Piramide.

[Google Scholar](#) ↗

[Rothman et al., 2003](#) R.B. Rothman, R.D. Clark, J.S. Partilla, M.H. Baumann

“(+)”-Fenfluramine and its major metabolite, (+)-norfenfluramine, are potent substrates for norepinephrine transporters”

J. Pharmacol. Exp. Ther., 305 (3) (2003), p. 1191-9, [10.1124/jpet.103.049684](https://doi.org/10.1124/jpet.103.049684) ↗

[Google Scholar](#) ↗

[Riva et al., 2017](#) P. Riva, A. Gabbiadini, L.J.R. Lauro, L. Andrichetto, C. Volpato, B.J. Bushman

Neuromodulation can reduce aggressive behavior elicited by violent video games

Cogn. Affect. Behav. Neurosci., 17 (2) (2017), pp. 452-459, [10.3758/s13415-016-0490-8](https://doi.org/10.3758/s13415-016-0490-8)

↗

[View in Scopus](#) ↗ [Google Scholar](#) ↗

[Scarpa et al., 2010](#) A. Scarpa, S.C. Haden, A. Tanaka

Being hot-tempered: Autonomic, emotional, and behavioral distinctions between childhood reactive and proactive aggression

Biol. Psychol., 84 (3) (2010), pp. 488-496, [10.1016/j.biopsycho.2009.11.006](#) ↗



[View PDF](#)

[View article](#)

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Scarpa et al., 2008](#) A. Scarpa, A. Tanaka, S.C. Haden

Biosocial bases of reactive and proactive aggression: the roles of community violence exposure and heart rate

J. Community Psychol., 36 (8) (2008), pp. 969-988, [10.1002/jcop.20276](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Schoorl et al., 2016](#) J. Schoorl, S. Van Rijn, M. De Wied, S.H. Van Goozen, H. Swaab

Variability in emotional/behavioral problems in boys with oppositional defiant disorder or conduct disorder: the role of arousal

Eur. Child Adolesc. Psychiatry, 25 (8) (2016), pp. 821-830, [10.1007/s00787-015-0790-5](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Schlüter et al., 2016](#) T. Schlüter, O. Winz, K. Henkel, T. Eggermann, S. Mohammadkhani-Shali, C. Dietrich, I. Vernaleken

MAOA-VNTR polymorphism modulates context-dependent dopamine release and aggressive behavior in males

Neuroimage, 125 (2016), pp. 378-385, [10.1016/j.neuroimage.2015.10.031](#) ↗



[View PDF](#)

[View article](#)

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Schlüter et al., 2013](#) T. Schlüter, O. Winz, K. Henkel, S. Prinz, L. Rademacher, J. Schmaljohann, I. Vernaleken

The impact of dopamine on aggression: an [18F]-FDOPA PET Study in healthy males

J. Neurosci., 33 (43) (2013), pp. 16889-16896, [10.1523/JNEUROSCI.1398-13.2013](#) ↗

[View in Scopus](#) ↗

[Google Scholar](#) ↗

[Sergiou et al., 2021](#) C.S. Sergiou, E. Santarnecchi, S.M. Romanella, M.J. Wieser, I.H. Franken, E.G. Rassin, J.D. van Dongen

tDCS targeting the Ventromedial Prefrontal Cortex reduces reactive aggression and modulates electrophysiological responses in a forensic population

Biol. Psychiatry.: Cogn. Neurosci. Neuroimaging (2021), [10.1016/j.bpsc.2021.05.007](#) ↗

[Google Scholar](#) ↗

- [Siep et al., 2019](#) N. Siep, F. Tonnaer, V. van de Ven, A. Arntz, A. Raine, M. Cima
Anger provocation increases limbic and decreases medial prefrontal cortex connectivity with the left amygdala in reactive aggressive violent offenders
Brain Imaging Behav., 13 (5) (2019), pp. 1311-1323, [10.1007/s11682-018-9945-6](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Skibsted et al., 2017](#) A.P. Skibsted, S.D. Cunha-Bang, J.M. Carré, A.E. Hansen, V. Beliveau, G.M. Knudsen, P.M. Fisher
Aggression-related brain function assessed with the Point Subtraction Aggression Paradigm in fMRI
Aggress. Behav., 43 (6) (2017), pp. 601-610, [10.1002/ab.21718](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Sollberger and Ehlert, 2016](#) S. Sollberger, U. Ehlert
How to use and interpret hormone ratios
Psychoneuroendocrinology, 63 (2016), pp. 385-397, [10.1016/j.psyneuen.2015.09.031](#) ↗
 [View PDF](#) [View article](#) [View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Song et al., 2020](#) J.H. Song, T. Colasante, T. Malti
Taming anger and trusting others: roles of skin conductance, anger regulation, and trust in children's aggression
Br. J. Dev. Psychol., 38 (1) (2020), pp. 42-58, [10.1111/bjdp.12304](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Stadler et al., 2007](#) C. Stadler, F.D. Zepf, L. Demisch, M. Schmitt, M. Landgraf, F. Poustka
Influence of rapid tryptophan depletion on laboratory-provoked aggression in children with ADHD
Neuropsychobiology, 56 (2-3) (2007), pp. 104-110, [10.1159/000112951](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Stevens et al., 2011](#) F.L. Stevens, R.A. Hurley, K.H. Taber
Anterior cingulate cortex: unique role in cognition and emotion
J. Neuropsychiatry Clin. Neurosci., 23 (2) (2011), pp. 121-125, [10.1176/jnp.23.2.jnp121](#) ↗
[View in Scopus](#) ↗ [Google Scholar](#) ↗
- [Stoppelbein et al., 2014](#) L. Stoppelbein, L. Greening, A. Luebke, P. Fite, S.P. Becker
The role of cortisol and psychopathic traits in aggression among at-risk girls: Tests of mediating hypotheses

Aggress. Behav., 40 (3) (2014), pp. 263-272, [10.1002/ab.21513 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

Taylor, 1967 S.P. Taylor

Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression

J. Personal., 35 (1967), pp. 297-310, [10.1111/j.1467-6494.1967.tb01430.x ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

Thomson and Beauchaine, 2018 N.D. Thomson, T.P. Beauchaine

Respiratory sinus arrhythmia mediates links between borderline personality disorder symptoms and both aggressive and violent behavior

J. Personal. Disord., 33 (4) (2018), pp. 544-559, [10.1521/pedi_2018_32_358 ↗](#)

[Google Scholar ↗](#)

Thomson et al., 2019 N.D. Thomson, K.A. Kiehl, J.M. Bjork

Violence and aggression in young women: the importance of psychopathy and neurobiological function

Physiol. Behav., 201 (2019), pp. 130-138, [10.1016/j.physbeh.2018.11.043 ↗](#)



[View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

Tuvblad et al., 2009 C. Tuvblad, A. Raine, M. Zheng, L.A. Baker

Genetic and environmental stability differs in reactive and proactive aggression

Aggress. Behav., 35 (6) (2009), pp. 437-452, [10.1002/ab.20319 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

Uddin et al., 2017 L.Q. Uddin, J.S. Nomi, B. Hébert-Seropian, J. Ghaziri, O. Boucher

Structure and function of the human insula

J. Clin. Neurophysiol.: Off. Publ. Am. Electroencephalogr. Soc., 34 (4) (2017), p. 300, [10.1097/WNP.0000000000000377 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

Van Bokhoven et al., 2005 I. Van Bokhoven, S.H.M. Van Goozen, H. Van Engeland, B. Schaal, L.

Arseneault, J.R. Séguin, R.E. Tremblay

Salivary cortisol and aggression in a population-based longitudinal study of adolescent males

J. Neural Transm., 112 (8) (2005), pp. 1083-1096, [10.1007/s00702-004-0253-5 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

- [Van Bokhoven et al., 2006](#) I. Van Bokhoven, S.H. Van Goozen, H. Van Engeland, B. Schaal, L. Arseneault, J.R. Séguin, R.E. Tremblay
Salivary testosterone and aggression, delinquency, and social dominance in a population-based longitudinal study of adolescent males
Horm. Behav., 50 (1) (2006), pp. 118-125, [10.1016/j.yhbeh.2006.02.002 ↗](#)
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [Van Donkelaar et al., 2020](#) M.M. Van Donkelaar, M. Hoogman, E. Shumskaya, J.K. Buitelaar, J. Bralten, B. Franke
Monoamine and neuroendocrine gene-sets associate with frustration-based aggression in a gender-specific manner
Eur. Neuropsychopharmacol., 30 (2020), pp. 75-86, [10.1016/j.euroneuro.2017.11.016 ↗](#)
 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)
- [Van Donkelaar et al., 2018](#) M.M. Van Donkelaar, M. Hoogman, I. Pappa, H. Tiemeier, J.K. Buitelaar, B. Franke, J. Bralten
Pleiotropic contribution of MECOM and AVPR1A to aggression and subcortical brain volumes
Front. Behav. Neurosci., 12 (2018), p. 61, [10.3389/fnbeh.2018.00061 ↗](#)
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [Vigil-Colet et al., 2015](#) A. Vigil-Colet, U. Lorenzo-Seva, F. Morales-Vives
The effects of ageing on self-reported aggression measures are partly explained by response bias
Psicothema (2015), pp. 209-215, [10.7334/psicothema2015.32 ↗](#)
[View in Scopus ↗](#) [Google Scholar ↗](#)
- [Vitaro and Brendgen, 2011](#) F. Vitaro, M. Brendgen
Subtypes of aggressive behaviors: etiologies, development and consequences
T. Bliesner, A. Beelman, M. Stemmler (Eds.), Antisocial Behavior and Crime: Contributions of Theory and Evaluation Research to Prevention and Intervention, Hogrefe, Goettingen (2011)
[Google Scholar ↗](#)
- [Vongas and Al Hajj, 2017](#) J.G. Vongas, R. Al Hajj
The effects of competition and implicit power motive on men's testosterone, emotion recognition, and aggression
Horm. Behav., 92 (2017), pp. 57-71, [10.1016/j.yhbeh.2017.04.005 ↗](#)

[View PDF](#)[View article](#)[View in Scopus ↗](#)[Google Scholar ↗](#)

[Von Polier et al., 2014](#) G.G. Von Polier, C.S. Biskup, W.F. Kötting, S. Bubenzer, K. Helmbold, A. Eisert, F.D. Zepf

Change in electrodermal activity after acute tryptophan depletion associated with aggression in young people with attention deficit hyperactivity disorder (ADHD)

J. Neural Transm., 121 (4) (2014), pp. 451-455, [10.1007/s00702-013-1119-5 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Wagels et al., 2018](#) L. Wagels, M. Votinov, T. Kellermann, A. Eisert, C. Beyer, U. Habel

Exogenous testosterone enhances the reactivity to social provocation in males

Front. Behav. Neurosci., 12 (2018), p. 37, [10.3389/fnbeh.2018.00037 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Wagner and Abaied, 2016](#) C.R. Wagner, J.L. Abaied

Skin conductance level reactivity moderates the association between parental psychological control and relational aggression in emerging adulthood

J. Youth Adolesc., 45 (4) (2016), pp. 687-700, [10.1007/s10964-016-0422-5 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Wagner and Abaied, 2015](#) C.R. Wagner, J.L. Abaied

Relational victimization and proactive versus reactive relational aggression: the moderating effects of respiratory sinus arrhythmia and skin conductance

Aggress. Behav., 41 (6) (2015), pp. 566-579, [10.1002/ab.21596 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Walters, 2005](#) G.D. Walters

Proactive and reactive aggression: a lifestyle view

J.P. Morgan (Ed.), Psychology of Aggression, Nova, Hauppauge, NY (2005), pp. 29-43

[Google Scholar ↗](#)

[Weidler et al., 2020](#) C. Weidler, U. Habel, P. Wallheinke, L. Wagels, L. Hofhansel, S. Ling, B. Clemens

Consequences of prefrontal TDCS on inhibitory control and reactive aggression

Soc. Cogn. Affect. Neurosci. (2020), [10.1093/scan/nsaa158 ↗](#)

[Google Scholar ↗](#)

[Weidler et al., 2019](#) C. Weidler, L. Wagels, C. Regenbogen, L. Hofhansel, J.A. Blendy, B. Clemens, U. Habel

The influence of the OPRM1 (A118G) polymorphism on behavioral and neural correlates of aggression in healthy males

Neuropharmacology, 156 (2019), Article 107467, [10.1016/j.neuropharm.2018.12.014 ↗](#)

 [View PDF](#) [View article](#) [View in Scopus ↗](#) [Google Scholar ↗](#)

[Weierstall and Elbert, 2011](#) R. Weierstall, T. Elbert

The appetitive aggression scale—development of an instrument for the assessment of human's attraction to violence

Eur. J. Psychotraumatol., 2 (1) (2011), p. 8430, [10.3402/ejpt.v2i0.8430 ↗](#)

[Google Scholar ↗](#)

[Werhahn et al., 2020](#) J.E. Werhahn, S. Mohl, D. Willinger, L. Smigielski, A. Roth, C. Hofstetter, D. Brandeis

Aggression subtypes relate to distinct resting state functional connectivity in children and adolescents with disruptive behavior

Eur. Child Adolesc. Psychiatry (2020), pp. 1-13, [10.1007/s00787-020-01601-9 ↗](#)

[Google Scholar ↗](#)

[White et al., 2013](#) S.F. White, S. Brislin, S. Sinclair, K.A. Fowler, K. Pope, R.J.R. Blair

The relationship between large cavum septum pellucidum and antisocial behavior, callous-unemotional traits and psychopathy in adolescents

J. Child Psychol. Psychiatry, 54 (5) (2013), pp. 575-581, [10.1111/j.1469-](#)

[7610.2012.02603.x ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[White et al., 2016](#) S.F. White, M. VanTieghem, S.J. Brislin, I. Sypher, S. Sinclair, D.S. Pine, R.J.R. Blair

Neural correlates of the propensity for retaliatory behavior in youths with disruptive behavior disorders

Am. J. Psychiatry, 173 (3) (2016), pp. 282-290, [10.1176/appi.ajp.2015.15020250 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Xu et al., 2014](#) Y. Xu, A. Raine, L. Yu, A. Krieg

Resting heart rate, vagal tone, and reactive and proactive aggression in Chinese children

J. Abnorm. Child Psychol., 42 (3) (2014), pp. 501-514, [10.1007/s10802-013-9792-2 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Yang et al., 2017](#) Y. Yang, S.H. Joshi, N. Jahanshad, P.M. Thompson, L.A. Baker
Neural correlates of proactive and reactive aggression in adolescent twins
Aggress. Behav., 43 (3) (2017), pp. 230-240, [10.1002/ab.21683 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Zepf et al., 2008](#) F.D. Zepf, C. Stadler, L. Demisch, M. Schmitt, M. Landgraf, F. Poustka
Serotonergic functioning and trait-impulsivity in attention-deficit/hyperactivity-disordered boys (ADHD): influence of rapid tryptophan depletion
Hum. Psychopharmacol.: Clin. Exp., 23 (1) (2008), pp. 43-51, [10.1002/hup.896 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Zhang et al., 2016](#) W. Zhang, C. Cao, M. Wang, L. Ji, Y. Cao
Monoamine oxidase a (MAOA) and catechol-o-methyltransferase (COMT) gene polymorphisms interact with maternal parenting in association with adolescent reactive aggression but not proactive aggression: evidence of differential susceptibility
J. Youth Adolesc., 45 (4) (2016), pp. 812-829, [10.1007/s10964-016-0442-1 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Zhang and Gao, 2015](#) W. Zhang, Y. Gao
Interactive effects of social adversity and respiratory sinus arrhythmia activity on reactive and proactive aggression
Psychophysiology, 52 (10) (2015), pp. 1343-1350, [10.1111/psyp.12473 ↗](#)

[View in Scopus ↗](#) [Google Scholar ↗](#)

[Zhang et al., 2018](#) D. Zhang, Y. Zhou, J. Yuan
Speech prosodies of different emotional categories activate different brain regions in adult cortex: an fNIRS study
Sci. Rep., 8 (1) (2018), pp. 1-11, [10.1038/s41598-017-18683-2 ↗](#)

 [View PDF](#) [View article](#) [Google Scholar ↗](#)

[Zhou et al., 2006](#) J. Zhou, X. Wang, L. Li, X. Cao, L. Xu, Y. Sun
Plasma serotonin levels in young violent offenders: aggressive responding and personality correlates
Prog. Neuro-Psychopharmacol. Biol. Psychiatry, 30 (8) (2006), pp. 1435-1441, [10.1016/j.pnpbp.2006.05.021 ↗](#)

[View PDF](#)[View article](#)[View in Scopus](#)[Google Scholar](#)

[Zhu et al., 2019a](#) W. Zhu, X. Zhou, L.X. Xia

Brain structures and functional connectivity associated with individual differences in trait proactive aggression

Sci. Rep., 9 (1) (2019), pp. 1-12, [10.1038/s41598-019-44115-4](#)

[Google Scholar](#)

[Zhu et al., 2019b](#) R. Zhu, C. Liu, T. Li, Z. Xu, B. Fung, C. Feng, L. Wang

Intranasal oxytocin reduces reactive aggression in men but not in women: a computational approach

Psychoneuroendocrinology, 108 (2019), pp. 172-181, [10.1016/j.psyneuen.2019.06.016](#)

[View PDF](#)[View article](#)[View in Scopus](#)[Google Scholar](#)

[Zijlmans et al., 2021](#) J. Zijlmans, R. Marhe, L. van Duin, M.J. Luijckx, F. Bevaart, A. Popma

No association between autonomic functioning and psychopathy and aggression in multi-problem young adults

Front. Psychol., 12 (2021), p. 686, [10.3389/fpsyg.2021.645089](#)

[Google Scholar](#)

[Zimmermann et al., 2012](#) M. Zimmermann, M. Grabemann, C. Mette, M. Abdel-Hamid, J. Ueckermann, M. Kraemer, F.D. Zepf

The effects of acute tryptophan depletion on reactive aggression in adults with attention-deficit/hyperactivity disorder (ADHD) and healthy controls

PLoS One, 7 (3) (2012), Article e32023, [10.1371/journal.pone.0032023](#)

[View in Scopus](#)

[Google Scholar](#)

Cited by (45)

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